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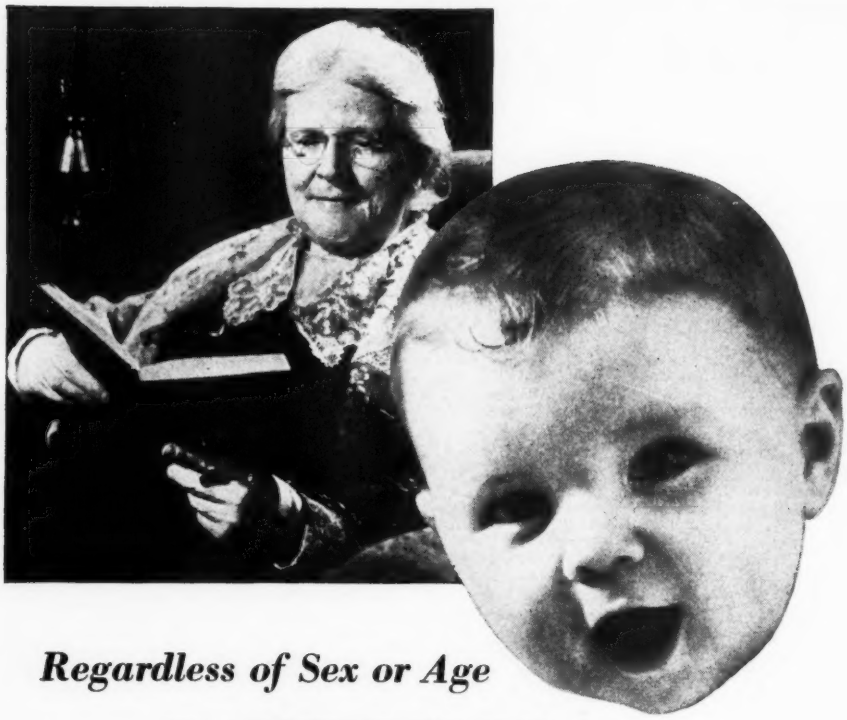

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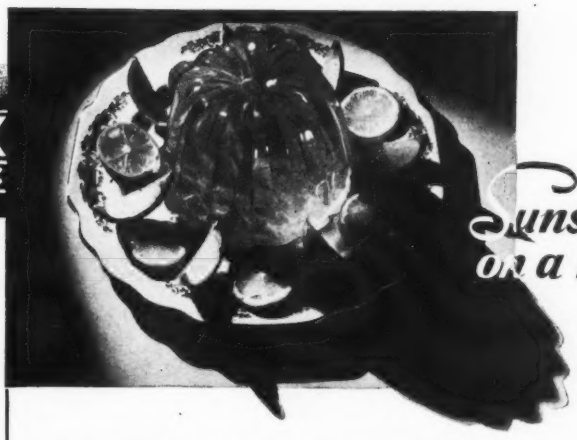

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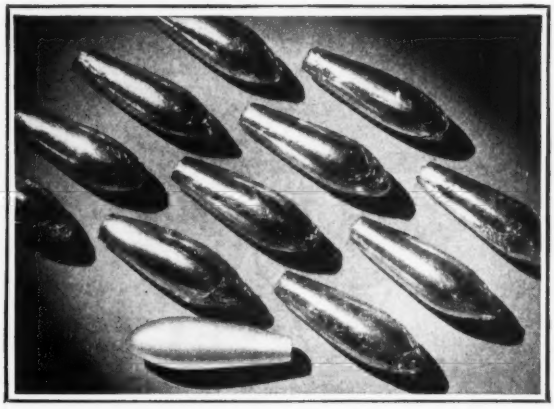

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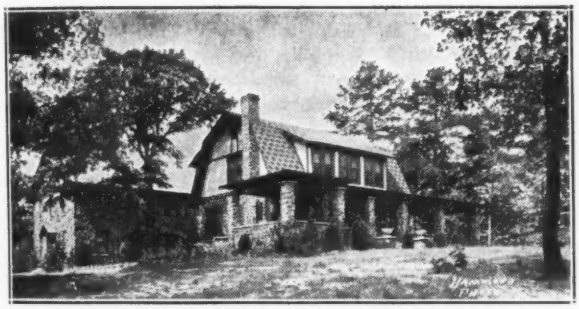

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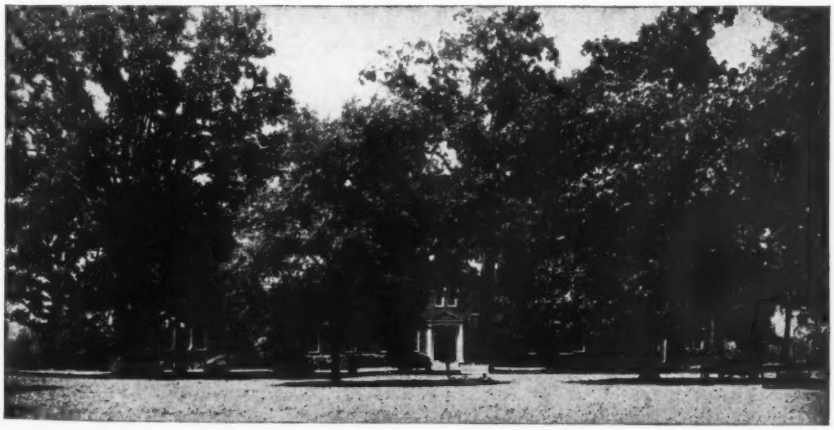

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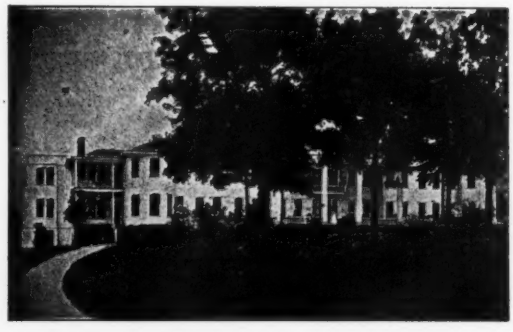

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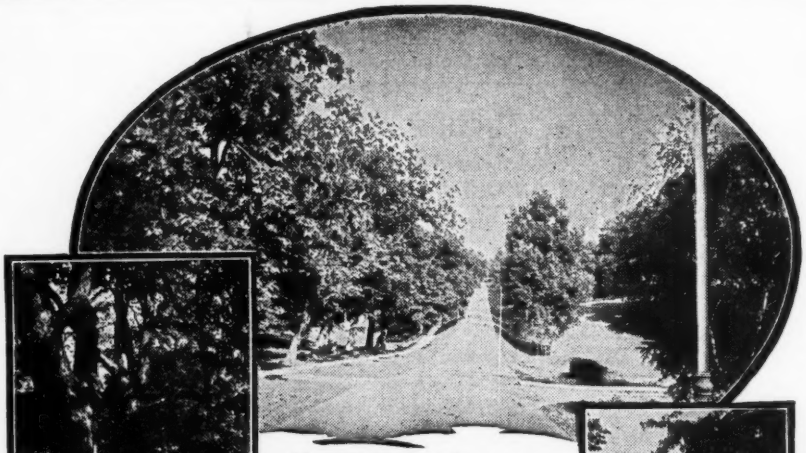

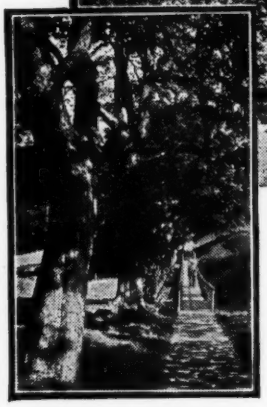

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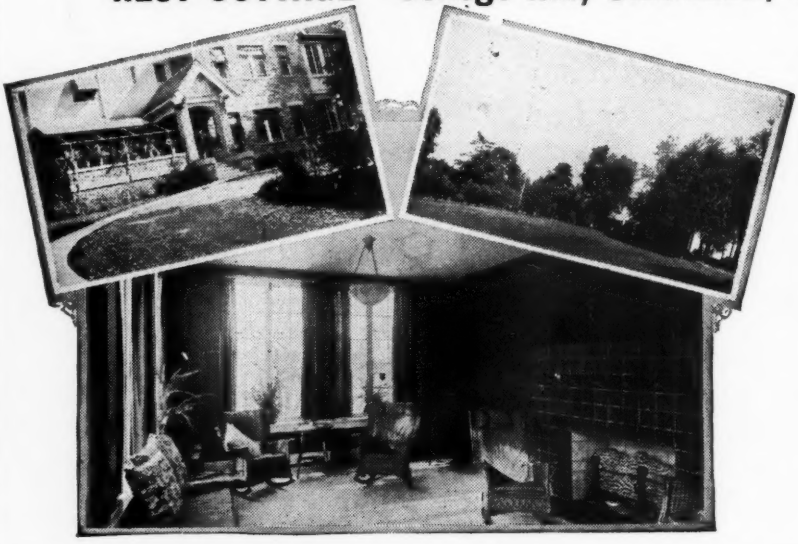

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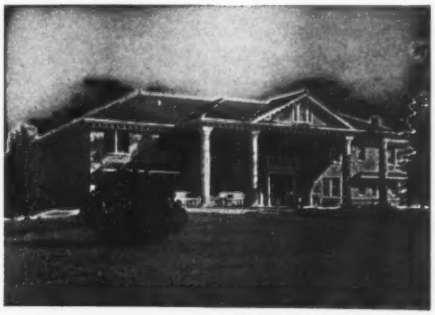

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VASCULAR CHANGES IN THE GLOMERULI OF THE KIDNEY AND THE EFFECT ON THE OTHER STRUCTURES OF THE NEPHRON*

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and
WILMA J. GREEN,‡
Oklahoma City, Oklahoma

No structure of vertebrate anatomy has been the subject of so many studies, nor the basis of so much speculation, as have been the glomeruli of the kidney. Ever since the beginning of histopathology great importance has been attached to the pathology of the glomerulus. So great has been the importance that has been attached to its lesions that the present classification of nephritis is based on glomerular pathology. Also, the current explanation of kidney function considers the glomerulus as the most important part of the kidney functional unit. Even hypertension is regarded by many essayists as being due to glomerular disease. But the whole truth as to the structure, function, or pathology of these minute structures has not yet been told. In fact, there is not an unanimity of opinion on any of these points. Therefore the report of another study is not out of place, and it is submitted in the hope that it will help to clear up some of the present doubt.

Thanks to the painstaking work of McGregor,[1] Vimtrump,[2] and the Bensleys,[3] we now have an accurate description of the structure of the glomerulus, and one which enables us to explain the abnormalities which have been found. A brief account of this structure is necessary to understand the pathology to be discussed in these studies.

According to these workers, the afferent glomerular artery on entering the capsule of Bowman, breaks up into from two to five primary branches. These in turn branch, and from these capillaries take off and run a tortuous course; they then unite to form arterioles, which unite to form a single or efferent artery, which leaves the glomerulus beside the afferent artery. This arrangement of vessels gives the glomerulus a lobed structure. The vessels entering and leaving the glomerulus, and those of the glomerulus itself have the same structure as vessels of the same caliber in other parts of the body. These workers also state that there are normally no anastomoses between the capillary groups or lobes, nor between any capillary and any other capillary within a lobe, nor between any part of a capillary and any other part of itself. The glomerular vessels are invested completely with a single layer of flat epithelium which is continuous with the epithelial lining of Bowman's capsule. This lobulation of the glomerulus, (*See Fig. 1*) the independence of these lobes, and the independence of the individual capillaries within the lobes, are important facts to be remembered in any discussion of kidney glomeruli, as will be shown later. It will be seen from the foregoing that glomerular disease might be expected to be vascular disease, at least primarily or basically.

Throughout the discussion of renal disease the importance of glomerular pathology has been recognized. Reasons for this importance have been stated, on presumptive evidence. The work of Zeckwer,[4] and Moore[5] has finally proven that at least one reason for the importance of glomerular disease is that all the blood going to the essential functioning tissues of the kidney passes through the glomeruli. In other words, the efferent glomerular artery supplies the tubule connected with a given glomerulus. This supply is also individual; that is, there are no normal

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†Professor of Pathology, University of Oklahoma School of Medicine.

‡Instructor in Pathology, University of Oklahoma School of Medicine.

Fig. 1
Intercapillary fibrosis, lobulation preserved, atrophy of dependent tubule.

anastomoses between the afferent, nor between the efferent glomerular arteries, nor between the tubular arteries or capillaries, of the different kidney units. Therefore, so far as circulation is concerned, the glomerulus holds a key position in the kidney functional unit, and each unit is complete and independent of all other units.

As was said before, a great many studies of autopsy material have been made. A great many animal experiments have been carried on controlling this and that factor in attempts to produce experimentally the lesions seen in human material, and to decide the question of the etiology and relation of these lesions to the alteration of kidney function. But it is not in the province of this paper to trace the character and results of all these studies, nor to follow the course of the ideas based on them as these ideas have been stated, denied, reaffirmed, and redenied, like tennis balls batted back and forth in a game. Such a procedure is very interesting, but as there are no definite agreements as yet there would be no point in such a review except for entertainment.

A great variety of pathological changes has been described. Based on these descriptions, nephritis has been divided into various kinds, and theories of urine production have been advanced. Unfortunately most of the observations have been made on isolated selections. Although the number of observations was great, and their character of the highest order, conclusions based on them cannot be accepted as final. In most cases the observations were too narrowly confined to one fraction of the kidney units so that the true significance of the lesion was not realized. In most of the experiments carried out, either the lesion produced was not identical with the human lesion, or the experimentors were so absorbed in a certain phenomenon that they did not appreciate all the phenomena with which they were confronted. But as was said above, from these observations and studies, definite changes have been described as the basis of different forms of nephritis, and of other diseases. Thus McGregor,[6] as the result of very thorough histopathological studies has described the glomerular lesion now generally considered to be the basis of essential hypertension.

But we who study the material that comes to our microscope from the autopsy table are continually confronted with exceptions and contradictions, cases in which we do not find the pathology that is supposed to accompany that form of disease with which the patient suffered before death. A woman, aged 31, whose kidneys weighed 95 grams each, and which were discovered at an abdominal operation thirteen years before to be reduced, did not have hypertension, although her kidneys were the typical sclerotic kidneys of hypertension. A woman 23 years old, the combined weight of whose kidneys was 42 grams had no known nephritis until her last illness. In the cases illustrated by *Fig. 2*, the glomeruli are sprinkled with hyaline masses, chronic focal glomerular nephritis, yet the condition was not suspected before death.

These facts led us to believe that a more extensive study of the glomerular lesions was necessary, and that some attention should be paid to the tubules of affected glomeruli. It was with the purpose of securing information con-

Fig. 2
Focal hyalinization, no atrophy of tubule.

cerning the extent of pathologic processes in glomeruli and the effect of these changes on the other parts of the functional units of the kidney that these studies were begun several years ago.

The material used was from human sources and was obtained from autopsies over a period of two years. All reduced kidneys, regardless of the disease or event causing death, were preserved for study. If the reduction was due to hydronephrosis, infarction, cicatrization following septic processes, hypoplasia, or other processes not dependent upon changes, the material was discarded as unsuitable for this problem. Further microscopic study showed that in some cases the reduction was due to medullary atrophy and other non-cortical lesions. This material was also discarded. There remained about a dozen cases with definite glomerular disease. The specimens obtained showed practically all previously described types of glomerular disease. Some of the series studied were from cases with marked hypertension, some were not so marked, and the rest were not clinically hypertensive cases. But strange to say, much more profound pathologic changes were detected in some of the cases in which the blood pressure was little if any at all above normal than in those with marked hypertension. Since these studies deal with pathological anatomy, no attempt will be made to correlate the finding with altered physiology. That is a problem in itself.

The serial section method was employed in these studies because only by this means can one examine a structure throughout its entire extent, obtain a true knowledge of its morphology and structure, and make out the extent of any change that may be found. The material secured was fixed in 4 per cent formaldehyde. Blocks of tissue were taken from several places in the kidney. These were mounted by the paraffin embedding method. The mounted material was cut in sections seven micra thick, thus sectioning all capillaries. The sections were mounted on numbered microscopic slides in the order in which they were cut from the block of tissue. In this way series were obtained of about thirty to sixty sections to a series. By having several series from each kidney it was possible to find out whether or not different changes occured in different parts of the same kidney. For the most part the series used in these studies were stained by the hematoxylin-eosin method. However, since McGregor claimed that a special anilin blue stain, that she had modified from Heidenhain's azan-carmine modification of Mallory's anilin blue, was the best for showing the beginning hyalinization of the capillary tufts, it was used on some of the sets studied. The microchemical test for amyloid was used in the cases in which hyaline degeneration occurred. This was done because of the great similarity in different kinds of hyaline when seen microscopically, to ascertain whether or not the hyaline was really an amyloid hyaline or a collagenous hyaline degeneration. These series were searched for glomeruli which showed vascular changes. Not all glomeruli in a given series showed the same degree of change, some none at all, others progressed so far that no idea of the original lesion could be made out. When a living glomerulus showing vascular change was found, it was traced through the series until all the lobes of the glomerulus had been studied. When the glomerulus had thus been studied in its entirety, other glomeruli in the same series were examined in the same way. In this manner all the glomeruli of a series showing vascular changes were studied to see whether or not other types or degrees of vascular change could be found.

The types of vascular changes found were as follows:

(A) *Extra-capillary Changes.*—(1) Intercapillary fibrosis which, like cicatricial tissue everywhere, by contraction, reduced the caliber of the capillaries and rendered them inelatsic so that very little blood could pass through.

(2) Hyperplasia and fibrosis proceeding from the capsule (*see Fig. 3*) forming crescents. This lesion compressed and caused atrophy of the lobes impinged upon.

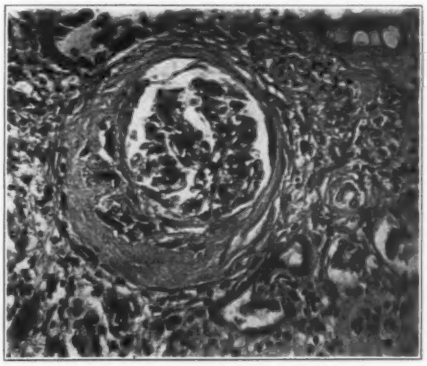

Fig. 3
Intercapsular crescent compressing glomerulus, atrophy of dependent tubule.

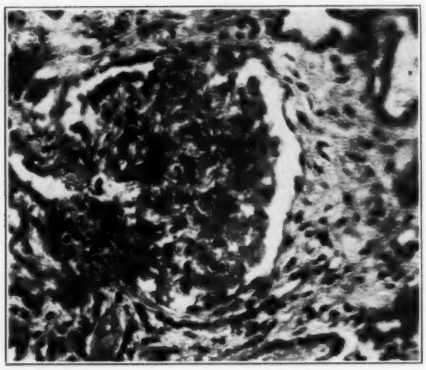

Fig. 4
Constriction of afferent artery, diffuse fibrosis, adhesion to capsule at two points, atrophy of dependent tubule.

(B) *Intracapillary Changes.*—(1) Swelling of the endothelium of some of the capillaries. This is a more or less focal lesion, probably temporary, or preceding some other form of lesion. This, while it lasted, closed the capillary affected.

(2) Hyaline degeneration.

(a) Collagenous degeneration preceded by a fibrosis and thickening of the capillary walls. The increase in thickness of the capillary wall was at the expense of the lumen, sometimes closing it entirely. This lesion usually involved an entire capillary or an entire lobe, sometimes a whole glomerulus, giving the picture seen in *Fig. 4.*

(b) Amyloid degeneration. This was usually focal at first, but finally involving the entire lobe or entire glomerulus as seen in *Fig. 6.*

(C) *Changes in the Afferent Vessel.*—(1) Perivascular fibrosis. This resulted in a narrowing of the lumen and inelasticity of the afferent vessel (*Fig. 4*).

(2) Hyaline degeneration of the vessel wall. For the most part this did not show the microchemical reaction of amyloid. Sometimes these changes in the afferent vessel were accompanied by some of the capillary changes enumerated above (*Fig. 4*).

In no case was calcification seen.

In all of these changes, unless the whole glomerulus was involved or the lumen of the afferent vessel reduced, there was evidence that a more or less adequate blood supply was passing through the glomerulus. This was shown by the caliber of the afferent and efferent arteries and the condition of the tubule part of the nephron. Thus the blocking of a capillary, or of a whole lobe of the glomerulus (the lesion of focal glomerular nephritis), while it evidently reduced the functional capacity of the glomerulus, had no appreciable effect on the nutrition of the tubule. The important point seemed to be not the type of lesion nor its extent, but whether or not an adequate blood supply reached the tubule. If it did not, whatever the glomerular lesion was or wherever it was situated, the tubule showed atrophy in proportion to the reduction of the blood supply passing through the glomerulus (*see Figs. 6, 4,* and *11*).

In a given kidney several types of vascular disease could be found in the glomeruli. Judging from the apparent age of these lesions it seemed that all glomeruli affected at a given time showed the same type of vascular change. At another time the insulting agent caused a different type of vascular change. So that the different types of vascular change depend on the causative factor.

In practically all the plates made for this paper there is a pathologic condition of the tubules that more or less confuses the picture, but is not connected with the disease shown in the glomeruli, except in the case of atrophy of the tubules. The tubule disease is an acute process due to a deleterious agent which acted shortly before death, while the pathologic condition of the glomeruli is subchronic or chronic.

In the series studied there were some sections in which there was amyloid degeneration in the glomerular tuft. An example of this is seen in *Fig. 5.* Amyloid degeneration involved some of the lobes of the glomerulus, but not all. It will

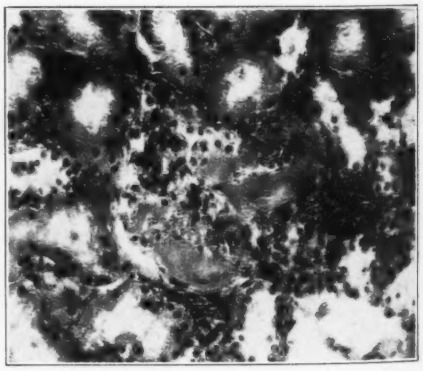

Fig. 5
Focal amyloid, no atrophy of dependent tubule.

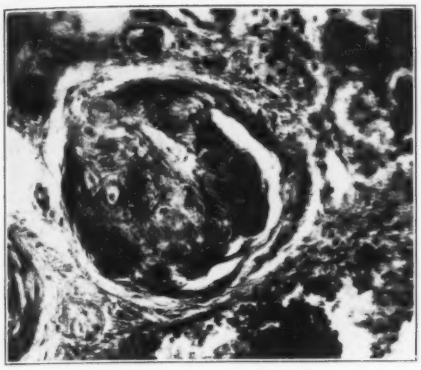

Fig. 6
Complete amyloid, complete atrophy of tubule.

be noticed that the lobes affected present a solid, homogeneous appearance that preserves the morphology of the lobes. No nuclei are seen in these areas, while other lobes of this same glomerulus are open and full of blood. The tubule that accompanies this glomerulus is not atrophied because the blood supply is able to go through the glomerulus and into the efferent vessel in sufficient quanities to supply the tubule. Practically every glomerulus studied in this case is in a condition closely resembling this one. Some of them have more amyloid degeneration, but the blood cells are found in the open vessels and the tubules are not atrophied. If the glomerulus is completely destroyed by the amyloid degeneration, as in *Fig. 6*, the glomerulus still retains its architecture, the lobes are still more or less separated and the glomerulus is not shrunken in size. Part of the subcapsular space still remains in one of the glomeruli. The capsule is thickened and the tubules are very atrophic. Some of the tubules are mere cords of atrophic epithelial cells, with no lumen whatsoever.

In other sections studied there was marked hyaline degeneration of the afferent vessel of the glomeruli. But after the afferent vessel divided into the capillaries of the glomerular lobes there was no discernible hyaline degeneration. There was some proliferation of endothelium, but as a whole most of the vessels in every glomerulus in this case were open as evidenced by the fact that they contained red blood cells and the tissue cells of the glomerulus were in a good state of nutrition. It will be seen that in *Fig. 7* there is hyalinization and thickening of the wall of the afferent artery, but that there is no narrowing of the lumen due to this degeneration. The capsule epithelium is not smooth and there seems to be the beginning of crescent formation, that is, the epithelium of the capsule is beginning to thicken and pile up to form a mass of epithelial cells in the subcapsular space. The tubular epithelium, although the site of cloudy swelling, is not reduced nor is the tubule diameter itself reduced.

In *Fig. 8* there is hyalinization and thickening of the wall of the afferent artery, but in this case there is narrowing of the lumen of the artery. This limits the inflow of blood to the glomerular tuft and as a result there are some loops of the tuft that are not open. In this case the capsule of Bowman is fairly smooth, but is thickened. The tubule of this nephron is

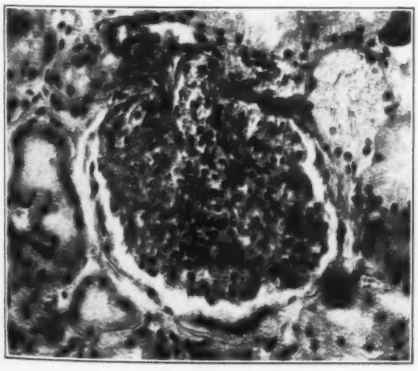

Fig. 7
Diffuse fibrosis, beginning crescent formation, sufficient blood passing through, no atrophy of tubules.

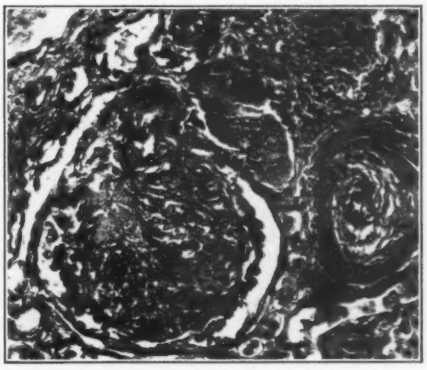

Fig. 8
Diffuse fibrosis, hyaline degeneration and constriction of afferent vessel, new periglomerular capsule forming which is covered by new epithelium.

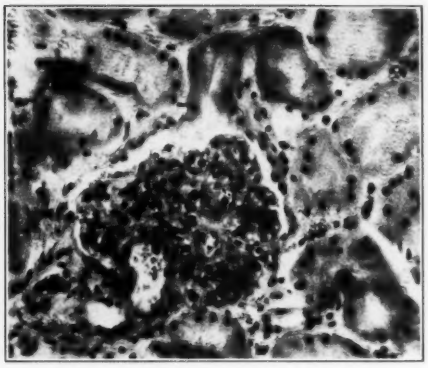

Fig. 9
Diffuse fibrosis, no constriction of vessel, no atrophy of tubule.

atrophied because the degree of fibrosis of the tufts was extensive and the narrowed lumen of the afferent artery reduced the blood supply to a very great degree.

In a glomerulus in which there is great proliferation of the connective tissue as well as the endothelium as in *Fig. 9,* but with the afferent artery in good condition with only slight beading of the endothelial lining and slight hyalinization, and with the lumen of normal diameter, there seems to be no effect on the tubules. Although the capsular epithelium is crinkled, the tubules are not in the least atrophied.

In a glomerulus in which there was great thickening of the capsule of Bowman, which was mostly hyalinized, the glomerular lobes were in a fair condition and open. The tubule is not smaller in size, but there is a slight atrophy of the epithelium, as will be seen in *Fig. 3.*

There were glomeruli in which the lumens of the afferent vessels were narrowed and in which the lobes were also fibrosed, but by following the circulation of the glomerulus through all the sections, it was found that some blood went through the glomerulus and entered the efferent vessel. In these cases, as in *Fig. 4,* the tubules were atrophied according to the amount of blood that was shut off. In this case the capsular epithelium was crinkled.

In some cases (*see Fig. 11*) the glomeruli were entirely fibrosed and hyalinized. There was no evidence that any blood went through the glomerulus. The glomerulus was firmly attached to the capsule of Bowman and the entire mass was shrunken to about two-thirds the normal size of the glomerulus. Blood vessels could be traced up to the very edge of the fibrosed glomerulus, but ended there. As a result of this no blood reached the efferent vessels and the tubules were very small with a very tiny lumen. In the fibrosed glomeruli seen in various sections studied, the glomeruli were much smaller than this one, an indication that the process was not so recent as the one in the plate. The tubules in these cases were mere cords of very atrophic epithelial cells.

In *Fig. 11* the glomerulus is itself enlarged in size. It will be noticed, however, that there is still subcapsular space and that the capsule is comparatively thin and smooth. This is a case of compensatory hyperplasia of both tubule and glomerulus in an effort to do the work of the

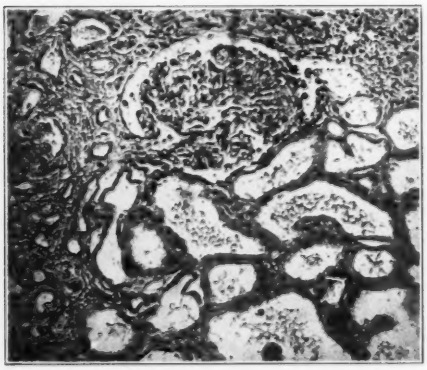

Fig. 10
This is a functioning nephron from a kidney weighing 95 gm. Some focal hyalinization, but otherwise fairly normal glomerulus. Tubules hyperplastic, epithelium normal.

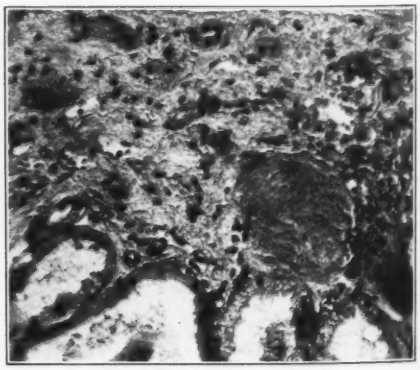

Fig. 11
Old completely fibrosed glomerulus. Tubule completely atrophied.

fibrosed glomeruli. The diameter of the tubule is greatly increased. The epithelial cells lining the tubule are of normal size, but the great size of the tubule gives the effect of atrophic epithelium. Around this tubule and glomerulus are other glomeruli and tubules that are greatly atrophied.

CONCLUSIONS

(1) Various kinds of vascular disease were found in the glomeruli of reduced kidneys.

(2) In some cases the lesions were focal, and although the foci were numerous, many capillary loops were still unaffected, allowing a sufficient blood supply to extra-glomerular structures. In some cases most or all of the loops were affected so that the blood supply to the extra-glomerular structures was reduced or completely cut off.

(3) The tubular lesion resulting from glomerular lesions was atrophy of the tubule epithelium.

(4) The effects of the glomerular lesions on the extra-glomerular structures was in proportion to the reduction of the efferent blood supply.

(5) The important point is not the kind but the extent of the glomerular lesions.

Whenever the blood was prevented from passing through the glomeruli, regardless of the cause, the tubules atrophied in proportion to the reduction of the blood supply. Clawson, Bell and Hartzell,[7] MacNider[8] and Lee-Brown[9] came to similar conclusions, although their work was undertaken for purposes other than ours, and their methods were entirely different.

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DISCUSSION (*Abstract*)

Dr. J. F. Pilcher, Galveston, Tex.—The understanding of histological changes in the kidneys has been confused by attempts to correlate function with morbid histology. Detailed and accurate anatomical studies such as this are necessary to a complete comprehension of kidney disease.

The concept that diminished blood flow through a glomerulus will affect the whole nephron is valuable and clears up the situation a great deal.

Dr. Charles Phillips, Temple, Tex.—Last year at our Richmond meeting, Dr. McCallum, of Johns Hopkins, read a paper before this Section on certain pathological changes in kidney glomeruli. Today Dr. Turley has given us another carefully prepared study. There is much in common in their findings and those of students of nephroses. I wonder whether some day we shall abandon our present complicated nomenclature of renal conditions and agree that living tissues are capable of a wide variation in reactions to stimuli. Investigations of this character, duration and quality are valuable to us all.

CHRONIC SUBDURAL HEMATOMA*

By C. C. NASH, M.D.,
Dallas, Texas

The pathology of this trouble has been understood since Virchow first described it in 1857. Many accurate descriptions of the gross appearance appeared in the literature prior to the discovery of the microscope. Prior to Virchow's description of the histology of the lesion little or nothing was known concerning the microscopical appearance of the clot. To this then well recognized disease of unknown etiology the name hematoma dura matris was given by Virchow. His description was so clear that practically nothing has since been added to its histopathology. The etiology was obscure and various authors had attempted explanations of the origin of the hematoma. The cases were all diagnosed postmortem and were all practically among the insane and chronic alcoholics. For this reason it was thought that the vessels of the dura and cortex were friable and that the hemorrhage was spontaneous and occurred at intervals, thus explaining the lamination of the clot which was sometimes seen. It was for this reason that Virchow argued that the lesion was not due to trauma, but to chronic inflammation of the dura. No cases were studied except those which came to autopsy and thus the name of pachymeningitis hemorrhagica was given to this entity by the pathologists and as such it was given a place in the textbooks of pathology and neurology.

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In 1923, Putnam,[2] working in Dr. Cushing's laboratory in the Peter Bent Brigham Hospital in Boston, carefully studied the histological appearance of a subdural blood clot. This clot had been removed from a boy of eighteen, who had received a blow on the head sufficient to render him unconscious about seven weeks before the operation. He noticed the similarity of the clot removed at operation to the appearance of clots removed at autopsy and compared the microscopical slides with several autopsy specimens. They were considered identical. Thus, for the first time it was definitely proven that pachymeningitis hemorrhagica occurring in previously healthy persons is due to trauma. Many articles have appeared in the literature bearing this out since the publication of Putnam and Cushing's work in 1925.

The situation of the hematoma in our series of eight cases was as follows: one in the frontal area (probably bilateral), one in the parieto-frontal area, two over the temporal lobe, and four in the parieto-temporal area. They were all proven unilateral by ventriculograms or encephalograms prior to operation except *Case 1.* In that case an encephalogram was made several months after operation, disclosing marked bilateral cortical atrophy.

The size of the cyst varies from very small up to several ounces in capacity. In *Case 4* of our series the cavity or depression in the cortex left by the removal of the hematoma held about 60 c. c. of normal saline solution. In two of our cases the sac had probably ruptured and spread the subdural space, making it very hard to gauge the amount of blood present.

The gross description of these cases is given as shining black, blue or greenish through the exposed dura. In our limited experience there was a thin layer of clot adherent to the dura, but no clot lying against the cortex except in *Case 7,* and the gross appearance was that the intact arachnoid formed the inner wall of the hemorrhagic cyst. In none of our cases was the clot a solid one, and all were soft and fragile, except in *Cases 1, 7* and *8,* and in each of these cases the trouble was of ten and eight months' and two and one-half years' duration, respectively. In all cases except these three the clot was easily wiped off the dura with wet cotton. In them the clot was firmly attached to the dura and was removed with the curette. Strange to say there was no oozing from the dura after the clot had been scraped away.

The etiology of the formation of the hematoma has never been satisfactorily explained. A more recent theory is that of W. James Gardner, who says:

"It is seldom possible to demonstrate the original source of the hemorrhage. The preponderance of evidence, however, would seem to indicate that the vascular rupture occurs in one or more of the cerebral veins as they cross the subdural space to enter the longitudinal sinus."

Leary, of Boston, in a recent issue of the *Journal of the American Medical Association,* says that in some cases of autopsy torn veins have been demonstrated.

Granted that this theory is correct, then the next stumbling block is, why is there frequently such a long interval from the time of injury until definite symptoms arise or why the latent interval?

Cushing and Putnam concluded that following the initial trauma there is a rapidly developing hematoma with the growth of capillaries in the hematoma wall and that some of these capillaries rupture and bleed into the hematoma, thus causing it to increase in size. This theory is confirmed by Leary in the paper previously mentioned.

Gardner, in 1931, advanced the theory that the wall of the hematoma became a dialysing membrane and proved by immersion of a hemorrhagic cyst in the patient's spinal fluid that the contents of the cyst increased 2.9 per cent in sixteen hours.

Zollinger and Gross (*Journal of the American Medical Association,* July 28, 1934) conclude:

(1) The wall of a subdural hematoma acts as a semipermeable membrane.

(2) Blood within a subdural hematoma breaks down slowly over a period of months.

(3) Disintegration of blood produces a great rise in its effective osmotic pressure.

(4) Blood within a subdural hematoma is progressively diluted.

It is believed that progressive liberation of hemoglobin from the erythrocyte stroma and subsequent disintegration of the hemoglobin molecule increase the osmotic tension in a hematoma sac over a period of months and that fluid is therefore gradually drawn in from the surrounding structures. The resultant augmentation in size of the lesion causes rising intracranial pressure and progression of clinical symptoms. The late onset of symptoms is therefore directly dependent on the slow disintegration of red blood cells encapsulated within the hematoma membrane.

SYMPTOMS

The symptoms are those of increased intracranial tension, namely: headache, dizziness, nausea, vomiting, convulsions, coma, choked discs, paralysis, and so on.

Headache may be very severe and may require large doses of morphine to relieve. In one case it was very severe and was unilateral, being localized on the side of the lesion. In all our cases there was a history of an initial severe headache. The onset may be sudden, but it is generally gradual and progressive in severity and is not relieved by spinal punctures.

Dizziness has not been very pronounced, but some patients have been unsteady or walked as if drunken.

Nausea and vomiting have been very pronounced in some cases. Projectile vomiting has not been seen, but was noted in the history of the case.

Convulsions were very severe in one case. They were general in nature. Jacksonian convulsions occurred in one case.

Coma was profound in three of our cases, came on gradually in two and followed a convulsion in the third case.

Choked discs are not always found, but when present are of great assistance in making a diagnosis.

Hemiplegia and other localizing signs are not always present, but a careful neurological examination may reveal some evidence of pressure on a certain part of the brain.

The diagnosis is not easy and all the means at one's command are sometimes necessary.

Spinal puncture reveals a clear fluid, but microscopically there may be crenated red blood cells. The pressure of the spinal fluid is increased. The presence of crenated red blood cells is thought to be suggestive of this condition.

Ventriculograms are invaluable in obscure cases. The ventricles are shifted or pushed toward the sound side. This is regarded as diagnostic in a case with a history of head injury presenting the above symptoms.

TREATMENT

The treatment is surgical and the details vary in different clinics. We have found that an opening in the skull sufficient to wash and suck out the old blood and clots is all that is necessary and large bone flaps are not needed. A rubber tissue drain is left in the site of the hematoma until the discharge is clear or has ceased. This usually requires about 48 hours.

Consciousness is usually rapidly restored following the removal of pressure and recovery has been complete in all of our cases that survived the ordeal.

Case 1.—R. W. I. was admitted to Parkland Hospital September 15, 1928. He was a white man, aged 60, a telephone operator, married.

He was struck by an auto and knocked down, and was brought to the hospital in moderate shock, but answered questions and complained of a severe headache. He was put to bed and treated for shock. His general condition remained good, but the severe headache continued. The blood pressure ranged around 140/90 and repeated urinalyses were negative. His condition remained about the same for four weeks. He suffered constantly with severe generalized headache. Repeated careful neurological examinations failed to show any localizing signs; however, about thirty days after injury a weakness of the left face, arm, and leg was noted, and a ventriculogram was done. This revealed that the posterior and descending horns of the right lateral ventricle were closed. A right subtemporal decompression was done and a large quantity of old blood was found in the subdural space and sucked away. A rubber tissue drain was left and drainage was profuse for about 24 hours. The stupor did not lessen but gradually became deeper, and he died of hyperthermia one week following operation. No autopsy was done.

Case 2.—J. W. was admitted to Baylor Hospital November 1, 1930, and discharged February 1, 1931. He was a negro man, aged 35, a porter. Ten months previously he slipped and fell on icy pavement, striking the back of his head. He was stunned momentarily, but continued his duties as a porter or orderly in Baylor Hospital. About one week after falling he noticed a rather persistent headache, which became more severe. Examination revealed double choked discs, a right facial weakness, weakness of the right arm, a bilateral Babinski, and ankle clonus. Spinal puncture revealed a clear fluid with 30 mm. of mercury pressure. The Wassermann was negative. He was dull, rather indifferent to his surroundings, and was disoriented at times. A diagnosis of a lesion of the left frontal area was made, a large osteoplastic flap was turned down, and a large subdural clot was found extending all over the left frontal lobe.

Case 3.—P. L. C., referred by Drs. T. C. Gilbert and A. J. Schwenkenberg, was admitted to the Methodist Hospital December 19, 1930, and discharged January 17, 1931. He was a white man, aged 34 years, a farmer.

His chief complaints were: severe headache, nausea and vomiting.

About six weeks previously he had been knocked out in a fight, two weeks previously he contracted a severe cold and on the following day developed a severe headache. This continued for about one week and he improved, but there was a return of the headache and his temperature went as high as 106°. The headache and temperature continued for two days and then abated

for twenty-four hours. There was then a return of the headache accompanied by moderate fever and this condition had continued until hospital entry. The day before entry there was vomiting of the projectile type.

Physical examination revealed a temperature of 98°, pulse 68, blood pressure 152/74, respiratory rate 18. Heart and lungs were normal. The right pupil was irregular in outline and smaller than the left. Neither reacted to light. There was slight nystagmus. The neck seemed slightly stiff. The reflexes were normal with probably increased right knee jerk. He gradually grew comatose without any noticeable change in the physical findings. The spinal fluid was clear and pressure 18 mm. of mercury. Wassermann was negative.

A diagnosis of frontal lobe abscess or subdural hemorrhage was made. The physical signs were very indefinite and ventriculograms were made. These films showed air only in the right ventricle and this ventricle displaced far to the right.

A subtemporal decompression was done on the left side. On opening the dura there was a profuse flow of old dark blood containing many small clots. This was sucked and wiped away with wet cotton until dry, a small rubber tissue drain inserted, and the wound closed. He rapidly improved, speech returned, and on the fourth postoperative day he was begging to get up. That night he had severe headache, became aphasic and stupid and had definite right-sided motor weakness. On the sixth day the wound was reopened. A bluish black mass could be seen in the upper angle and more bone was rongeured away, exposing the lower part of the hematoma. It was opened and approximately 60 c. c. of thin black blood were sucked out. There was a thin layer of clot attached to the dura and a large depression was left in the cortex. The clot was removed by dull curette and wet cotton, a rubber tissue drain was inserted and the wound closed with fine black silk. Following this, convalescence was uneventful, the aphasia and muscle weakness rapidly cleared up and he was discharged on January 17, 1931, as cured.

Case 4.—I. S. was admitted to St. Paul's Hospital December 16, 1930, and discharged January 18, 1931. A traveling salesman, aged 63, he was referred by Drs. Jablow and Schwenkenberg. About six weeks previously he was injured in an automobile accident, receiving a small scalp wound in the frontal region, and he was dazed for a short while, but was not unconscious. He was off from work for two weeks and returned to the road, but with some headache. The headache became worse and he began to stagger. On December 13 he noticed weakness in the left hand and the following day was found unconscious in the bathroom. On entering the hospital there was weakness of the left leg and arm, headache was very severe, and he slept a great deal, was semistupid, vomited and had persistent hiccough. The pupils were slightly irregular, but reacted to light. The disc margins were blurred. The deep reflexes were increased in the left leg and arm. The abdominal and cremastetric were absent. There was a left Babinski. At times he seemed slightly confused.

The Wassermann was negative.

Urinalysis showed specific gravity 1.023, no albumin, sugar nor casts; acetone and diacetic acid 2 plus.

Four days after admission he lapsed into a stupor. An x-ray film of the head showed a linear fracture in the left parietal bone near the occipito-parietal suture.

I saw the patient on December 22, at which time he was in deep coma with definite paralysis of the left arm and leg. The pulse was 100, regular and full. The temperature was 98.4 and respirations were 24, shallow and irregular. The blood pressure was 116/80. From the history it was very evident that we were confronted with a progressive condition and in view of recent trauma and the paralysis of the left arm and leg, a diagnosis of chronic subdural hematoma was made and an operation advised. During the night he was given 50 grams of glucose intravenously at six-hour intervals, and was operated upon the following day. A right subtemporal decompression was done. The exposed dura was very dark and a small nick revealed the typical dark blood of a chronic subdural hematoma. There were many minute clots. The opening was enlarged and the cavity thoroughly washed out and sucked dry through wet cotton. The brain was markedly depressed and the cavity was estimated to have contained 60 c. c. of blood. A thin filmy clot was adherent to the dura and was easily removed with wet cotton. There was no clot on the arachnoid side of the cavity. A rubber tissue drain was inserted into this cavity and the muscle, fascia, galea and scalp carefully closed with interrupted fine black silk. Progress thereafter was slow but steady, and one week from the day of operation he was perfectly conscious and his memory clear. He was discharged from the hospital January 18, 1931. This patient has been seen many times since his discharge and is working steadily at his former position, that of a traveling salesman.

Case 5.—R. P. was admitted to St. Paul's Hospital October 30, 1931, and discharged November 15. He was a white college student, aged 21, referred by Dr. T. C. Gilbert.

This young man sustained an injury to his head in football practice about four weeks previous to his admission to the hospital. There had been moderate headache following the injury, which gradually increased in severity. The day previous to admission he complained of severe headache on arising from bed and while dressing himself fell and had a severe generalized convulsion. Following the convulsion, coma was profound and continuous and he was brought to Dallas in this condition. Shortly after admission there was a severe generalized convulsion. A careful neurological examination revealed no distinct localizing signs, though it was thought that the left face and left arm were more involved than the rest of the body. Spinal puncture revealed a clear fluid with a pressure of 20 mm. of mercury. Blood count showed a white count of 14,000, with 70 per cent polymorphonuclears.

There was involuntary passage of urine and feces. During the night there was another generalized convulsion and the following morning his condition was decidedly worse, the pulse being fast and weak, and respiration somewhat Cheyne-Stokes in type. His condition was getting worse and as the left side seemed involved more than the right, a right subtemporal decompression was advised. This was done the morning following admission. The brain was very tense and under great pressure. Some bloody fluid was encountered, a rubber tissue drain was placed over the frontal area and the wound closed in the usual manner. There

were no more convulsions and the patient was quiet. About forty-eight hours after the operation there was a sudden escape of dark blood into the dressing and the wound was inspected for signs of hemorrhage, but none was found. The following morning the patient was conscious and thereafter his convalescence was rapid.

This case was not absolutely proven to be a subdural hematoma, yet I am sure from the sudden drainage into the dressing of a large amount of bloody fluid containing many small clots that it was such a case. The rapid and spectacular recovery following is further proof.

Case 6.—John W., aged 27, a medical student, was injured October 8, 1932, about 6:00 p. m. When going upstairs he slipped and fell, striking the back of his head on the bannister. He was dazed for a few minutes, but was never unconscious. He had difficulty in getting to sleep, but after he fell asleep, slept soundly through the night. The following morning he awoke with headache and vomiting. By noon the nausea and vomiting had subsided, but headache continued. The headache was continuous, became progressively worse and he was very dizzy. He continued in school, but had no appetite and came to see me on October 12. His complaints were headache and dizziness and anorexia. A neurological examination was entirely negative, except for positive Romberg. X-ray films of the head were negative for fracture. He was ordered to bed with rigid restriction of fluid intake and magnesium sulphate four times daily. His condition became worse and two days later he was sent to Baylor Hospital, where a spinal puncture was performed. The fluid was clear with a pressure of 16 mm. of mercury and microscopically a few red cells. The headache was not improved by the spinal puncture. Dehydration, rest and icebags to the head were continued, but the headache became gradually more severe. Four days after admission he developed a paresthesia of the right face and right arm. Neurological examination revealed weakness of the right face and arm. The fundi were normal. Daily spinal punctures were done for several days, but with no relief of headache. At this time Dr. Schwenkenberg saw the patient and confirmed the above findings. In view of these findings, and particularly of the fact that his headache was never relieved in the slightest degree by spinal puncture and reduction of the pressure to one-half of the reading, it was decided that an encephalogram should be made. This was done on October 22 and the films showed an incomplete filling of the left lateral ventricle. A diagnosis of subdural hematoma was made and he was operated upon the following day. There was no aphasia, though at times he seemed to hesitate slightly in naming objects. Under general anesthesia a vertical incision was made in front of the left ear, the skull opened with a perforator and the opening slightly enlarged with a rongeur. It was noted at the time that bleeding from the scalp was greater than normal. The dura was noted as being very dark and a small nick was made in it. There was an immediate escape of a large amount of dark liquid blood. The sylvian veins were unusually large and in enlarging the dural opening a large vein was cut or torn. The bleeding could not be controllel with the electrosurgical current and finally a muscle graft had to be taken from the calf of the leg and applied to the bleeding point. This effectually controlled the bleeding, but he had lost so much blood that he was given a 500 c. c. transfusion of whole blood.

Motor aphasia was complete for forty-eight hours and gave us great concern, but at the end of this time began to clear up and speech was normal by the end of the week. The wound oozed considerably for forty-eight hours, but thereafter cleared and skin sutures were removed on the third day. The headache was gone when he aroused from the anesthetic and has never bothered him since. He has just completed his junior year in medicine.

Case 7.—Miss R. C. was admitted to Methodist Hospital November 20, 1933, and discharged December 29. She was 38 years old, white, single, a secretary. She had received a blow on the head, but did not lose consciousness, in an auto accident eight months previously. There was continuous headache afterward. Five days before admission, while she was standing putting on her hat about 9:00 a. m., she was seized with sudden very severe pain in the left side of her head, radiating from front to back, but centered chiefly just above the ear. The pain was so severe that she could not stand and soon after lying down she began to vomit. Several hypodermics of morphine were given to alleviate the pain, but only slight relief was obtained. Vomiting had been continuous and she had been unable to retain food or water. A neurological examination revealed a suggestion of left facial weakness. The head could be rotated normally to the right, but not to the left, and in attempting to do so she leaned the head on the left shoulder. The knee jerks were hyperactive with a tendency to clonus. No pathological reflexes were noted. There was nuchal rigidity, but no Kernig. The fundi were clear. Spinal fluid was clear, with a pressure of 12 mm. of mercury. On this history of a moderately severe head trauma, a diagnosis of chronic subdural hematoma was made. Ventriculograms showed a pronounced shift to the right. A small osteoplastic flap was made on the left. The dura was opened with base upward and a large thick, firm clot was found densely attached to the dura. There was a large amount of dark liquid blood in the subdural space outside of the clot. The clot was curetted, wiped and sucked away and, as far as it was possible to do so, all the old blood was aspirated out of the subdural space. A rubber drain was placed in the subdural space in the middle fossa. There was profuse drainage for twenty-four hours, which thereafter became less, and the drain was removed on the fourth postoperative day. She was discharged forty days after admission, entirely free from headache. Microscopic study of the clot showed organization with fibroblastic cell proliferation.

Recently this patient has had some three severe generalized convulsions. Encephalograms are normal.

Case 8.—J. D. R., aged 57, a farmer, was admitted to Baylor Hospital August 26, 1934, and discharged September 10. He was referred by Dr. Geo. L. Carlisle.

For the past three years he had had gradually increasing headache and during the past year he had been forgetful. About three months previously he was knocked down by a bull and received a blow on the head, but was not unconscious. The headache was much worse afterward and about two weeks before I saw him the right arm became weak and he dragged

Ventriculogram, Case 8. Ventricle shifting in subdural hematoma.

the right foot and staggered. During the previous ten days there had been gradually increasing stupor, and it was difficult to arouse him.

He could be aroused, answered questions slowly and obeyed commands. Sensory aphasia was present. The right arm was almost completely paralyzed, and the right leg was weak. The right biceps was exaggerated. The knee jerks were very weak and the ankle jerks absent. There was right facial weakness. The pupils were small, equal and responded to light. Visual fields were normal grossly. The discs were slightly swollen and the retinal veins were full. Spinal puncture revealed a clear fluid and a pressure of 18 mm. of mercury.

Blood and spinal fluid Wassermann were negative.

Ventriculograms revealed a marked shifting to the right of the ventricles and the pineal body.

A left subtemporal decompression was done and a large encapsulated subdural hematoma was found and easily removed without rupture. The dura was not discolored by the hematoma. Improvement was very striking and ten days after operation the patient was able to recall that in April, 1932, he sustained a rather severe head injury when a cultivator turned over on him, and that his headache followed this injury and had been continuous to the present illness.

Unfortunately the specimen was lost and histological study was not made.

SUMMARY

Chronic subdural hematoma is not an uncommon condition and should be thought of in patients with suspected brain tumor who give a history of preceding trauma.

The trauma in our series of cases has been of a rather mild degree and has not always been followed by temporary unconsciousness or loss of memory.

The symptoms may come on several months after the initial injury.

In comatose patients it may not be possible to elicit a history of trauma until after the patient has been operated upon and consciousness restored.

In our small series of cases, the seven who survived have had complete relief of symptoms and five have been completely restored to their former status.

DISCUSSION (*Abstract*)

Dr. James W. Brown, Houston, Tex.—We can diagnose most subdural hematomata clinically, but we run into difficulties at times. These patients may show a latent interval or may not. The symptoms may come on immediately after the accident or weeks or months later. Signs are sometimes practically absent. Some of these patients improve; and doubtless some of them heal without treatment, as cases have been reported in the literature. Most of them are fatal without operation, however. When there is a question of the presence of hematomata, I think we are justified in doing an exploratory operation or a pneumogram. The latter is not always satisfactory because the subdural hematomata may be of great extent and very thin. It seems that relatively mild injuries cause most hematomata. I have never seen a cerebellar hematoma diagnosed ante mortem, and I do not recall having seen one reported in the literature.

Few people see as many of these cases as Dr. Nash has, because they fail to recognize them or do not perform an operation or autopsy upon them. I fail to see why we should differentiate chronic subdural hematomata and acute subdural hematomata. The chronic ones are, as a rule, cases that have been unrecognized or untreated early. The essential nature of the condition is not changed by the passing of time, but secondary modifications do occur. A patient who dies within a week from subdural hematoma cannot be said

to be suffering from a chronic condition. Dr. Nash has mentioned the various methods of treating these hematomata. Some surgeons advocate operating upon them through one or two trephine openings. Others operate upon them by turning a flap, and still others operate as Dr. Nash does, through subtemporal decompression openings. I think that the latter method is by far the most satisfactory in practically all cases. The procedure can be carried out without much shock to the patient and gives enough exposure to explore for and remove subdural hematomata. I have never seen the trephine method used, but one certainly cannot get very good exposure by it, and some of the hematomata are too solid to be washed out. Should hemorrhage be caused by the operator's using this method, he would find great difficulty in controlling it. Turning of a flap is a larger operation which takes longer and is harder upon the patient. There is some tendency for postoperative hematoma formation beneath any flap. There are, of course, a few recurrent cases where a flap is indicated to stop a persistent hemorrhage. I believe that a bilateral subtemporal decompression should be used in all cases, more especially in those who have not had pneumograms, as subdural hematomata are very often bilateral and are occasionally on the side opposite to that to which the neurological signs point. It is unnecessary to remove the whole membrane or even the whole hematoma. Dr. Elsberg has demonstrated this point upon numerous occasions. Drainage should be done for 24 or 48 hours to prevent collection of fluid in the old cyst region and to allow the brain to fill the space formerly occupied by the hematoma. The decompression helps to take care of ensuing edema which is usually present.

Dr. Arthur J. Schwenkenberg, Dallas, Tex.—It was my privilege to have seen several of these cases with Dr. Nash. I wish to say a few words from the diagnostic standpoint. The neurological signs are strikingly those of an extracerebral lesion and, consequently, localizing signs do not manifest themselves until the hematoma has reached fair size. In practically all the cases there was localized tenderness of the skull at the site of the clot. In the elderly Jewish patient it was easy to regard the case as one of cerebral thrombosis. A careful history, however, revealed the fact that six weeks before, he had a slight injury to the head and there was a small scar above the right eye. X-ray examination revealed a small fracture in the left occipital region. There was no evidence of increased intracranial pressure, either with spinal measurement or examination of the eye grounds, although for ten days he was in a deep stupor and vomited a great deal. On pressure over the right frontoparietal region he constantly evinced pain. I decided that the lesion was extracerebral and, consequently, felt that a hematoma must be present. At operation Dr. Nash removed a large hematoma, which he has described.

As Dr. Nash has mentioned, many of these cases followed mild injuries with practically no disturbance in consciousness. I think there are many more cases than we realize. The case of the medical student demonstrates that only a slight injury is necessary. This patient merely slipped and fell down a few steps, striking his head. He continued in school, but a left frontoparietal headache became more intense. At examination he had a slight weakness of the right side of his face, a weakness of the right arm and a right Babinski. There was definite tenderness of the skull over the left rolandic area. After removal of the clot, recovery was uneventful.

The last case I did not see, but his recovery, I understand, was almost miraculous. It certainly demonstrates the importance of a careful history. The diagnosis was made by a very capable internist who appreciates the value of a good history.

THE ANATOMY OF THE PROSTATE AND VESICAL NECK*

By REX E. VAN DUZEN, M.D.,
Dallas, Texas

There is a wide difference of opinion concerning the merits of transurethral resection of the prostate and it has occurred to me that we might better understand what we are attempting to do if we review the embryology and anatomy of this region.

There are three contributions to medical literature that every urologist should read, namely: Lowsley, "The Human Prostate in Youth" (*Medical Record*, September 5, 1915); Tandler and Zuckerkandl, "Die Prostatahypertrophie;" and Randall, "The Human Prostate." These papers are most excellent. Lowsley, in 1912, described the embryological development of the prostate and it has never been revised. There are several steps in the development that I feel should be emphasized and our findings vary somewhat, especially in the formation of the lobes.

Lowsley described five groups of glands which he feels give rise to five lobes of the prostate (*Fig. 1*). I doubt that this is true, as our later slides fail to show such lobes.

In the eight months embryo (*Fig. 2*), we note the downward growth of the glands with tendency to branching and tortuosity of the ducts. At this stage the female embryo shows regression of the glands. It should be noted that the glands grow toward the external wall of the urethra and not toward the bladder.

In a four-year male (*Fig. 3*), we see further branching of the glands and they have become racemose. However, there is no attempt at encapsulation and the muscle of the internal sphincter is well developed.

In a twenty-year old male, unmarried (*Fig. 4*), the prostatic glands comprise a small part of

*Read in Section on Urology, Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

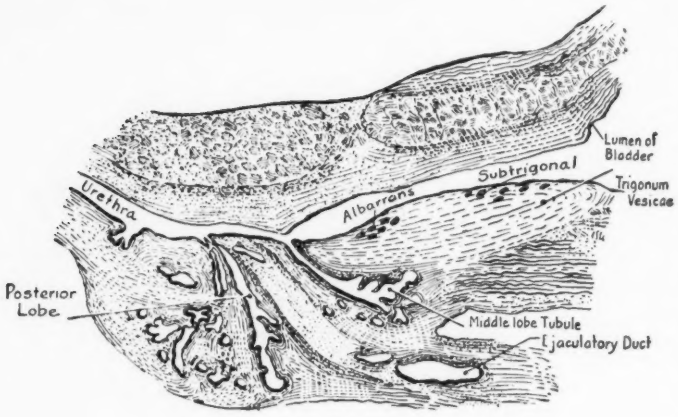

Fig. 1
Origin of five prostatic lobes, according to Lowsley.

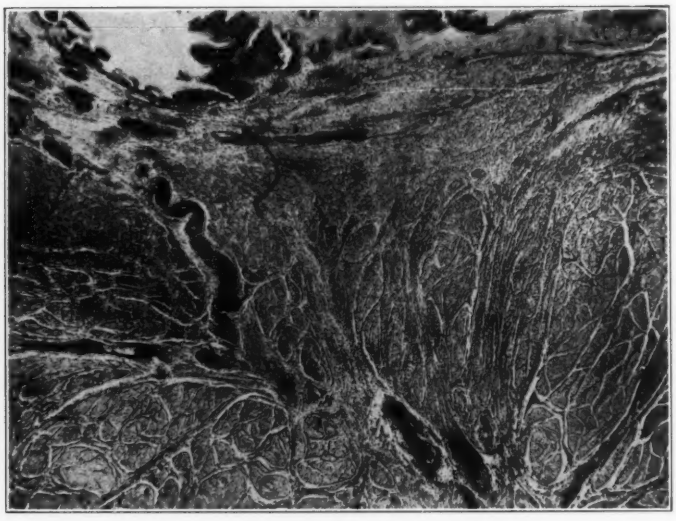

Fig. 2
Eight months embryo.

the urethral wall compared to later specimens. Both the so-called posterior and anterior lobes show well developed glands. However, it should be noted that the muscular wall about the internal sphincter has developed more than the glands.

In a twenty-eight-year old married man (*Fig. 5*), we see marked development of the prostatic glands compared to earlier specimens. But it should also be noted that the muscular wall separates the glands from the bladder, not a fibrous capsule. We must conclude that the glandular proliferation represents response to physiological activity.

In cases of early hypertrophy of the prostate (*Fig. 6*), we note the increase in size of the suburethral glands and increase in the fibrous tissue, especially in the region of the internal sphincter. The large prostatic glands still show the same arrangement as in earlier specimens. The left hemisection has been bisected and shows that in a lateral plane the glands extend to the periphery of the prostate, but there is no extension up toward the bladder.

Fig. 7 represents a moderate hypertrophy of the prostate gland. We should note this occurs at two places. We see the spheroids in the true prostatic glands and also in the suburethral glands. The arrangement of ducts and glands is not as distinct as in earlier specimens.

Fig. 8 represents the large hypertrophy. The glandular arrangement is entirely lost. It is from cases of this type that we could easily conclude that the prostate is an encapsulated organ, but these septa are areas of compression.

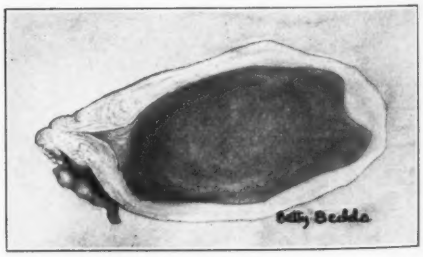

Fig. 3
Child, four years old.

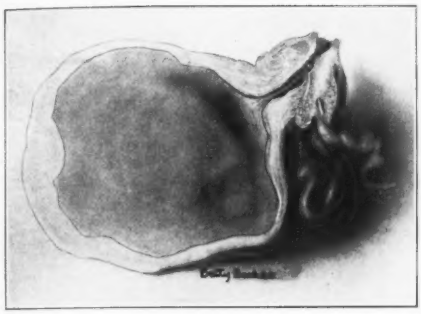

Fig. 4
Unmarried, twenty years old.

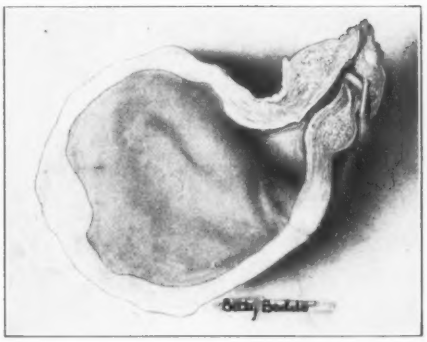

Fig. 5
Married, twenty-eight years old.

What is the prostate? Anatomists have pointed out that the prostate is not separated from the bladder by a fibrous capsule. Our specimens show that the bladder muscle is continuous with the prostatic muscle and the glands start, in the main, from about the verumontanum and grow outward, but only late in life backward toward the bladder. All the evidence points to the fact that the prostate is a differentiated portion of the urethra and not a separate anatomical structure. I like to compare the prostate with the pylorus of the stomach. We see highly developed glands in both structures. We never speak of the pylorus as a gland and why consider the prostate as an anatomical entity? It is true it has a physiological function foreign to the bladder and urethra. But in treating hypertrophy of the prostate, we must consider it as a part of the bladder and urethra and not as a physiological structure. Our problem is entirely anatomical because its enlargement or fibrous change may cause obstruction to the urinary flow. The writers of the past have followed Lowsley's description of the five lobes of the prostate, namely: the two lateral, the median, the anterior and the posterior. If we recall that throughout life, Bell's muscle compresses the floor of the urethra each time the internal sphincter is opened, it is easily seen that compression atrophy would develop and glandular development is more

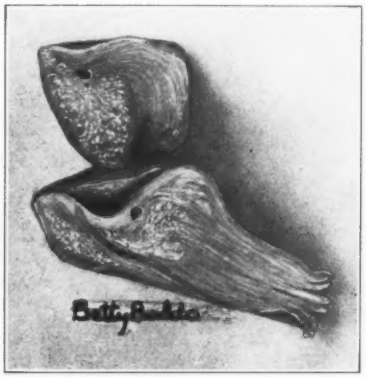

Fig. 6
Early hypertrophy.

Fig. 7
Senile hypertrophy of prostate.

Fig. 8
Large hypertrophy.

liable to take place at points of less pressure. Muscle fibers are found completely to encircle the urethra at all levels and no true septum can be demonstrated. I have already pointed out that the ejaculatory ducts give the impression of separating the posterior glands from the remainder, but more oblique sections show no septa. When these facts are considered, we must realize there is no division into lobes. The glands are first simple glands and later become racemose. They show a marked increase in size with onset of sexual activity. This is entirely physiological. At about 45 years of age we note that the suburethral glands, which have been very poorly developed, begin to increase in size. What does this indicate? Is it nature's attempt to compensate for exhaustion of the true prostatic glands? At the same time there is an increase in fibrous tissue, especially about the internal sphincter. Serralach has produced a similar fibrosis in the prostate of dogs which have been subjected to repeated masturbation. He feels that in the human, prolonged or incomplete intercourse, erotism without gratification, withdrawal, and so on, are predisposing factors. A French author, several years ago, pointed out that it was impossible to ejaculate and urinate at the same time. Is this fibrosis, the result of the repeated muscular contraction of internal sphincter during sexual excitement? I would doubt this. Involuntary muscle hypertrophies when it is overworked. Fibrosis usually means infection or toxic irritation. Recently I have resected a hypertrophic internal sphincter in a woman who always had from eight to twenty ounces of residual urine. There was no fibrosis. Marion has reported fibrosis, but I suspect his cases have infected bladders while, fortunately, my case did not.

Is Serralach correct that erotism predisposes to exudation into the muscular wall of the urethra? Are the sperm cells toxic if retained? If that is true, ligation of the vas should be performed for prevention of hypertrophy. Is the prostatic fluid toxic if retained? If either is toxic, I would suspect it is the prostatic secretion because we have all seen the muscular relaxation follow a prostatic massage. These questions all should receive more study.

We have been discussing changes in the prostate that still retains its normal architecture. The next change is in the glands themselves. Shortly after the increase in fibrosis and increase in size of the suburethral glands, we note that a few of the acini of the prostatic glands form adenomatous areas or spheroids. These gradually increase in size until the glandular tissue is entirely involved and then we see the large hypertrophies. What has produced this adenomatous change? Have the ducts become occluded by the exudation or by new formed fibrous tissue? We know that so-called hypertrophic glands yield little or no secretion on massage? We also see dilated acini in the microscopic slides which suggest retention. Is Ballenger correct that prostatic hypertrophy may be prevented by routine prostatic massage after forty-five? When the hypertrophy reaches the size where it extends into the bladder, the trigon muscle at first hypertrophies and later atrophies. The internal sphincter is often replaced by a fibrous inelastic structure which occasionally may extend downward and involve the external sphincter to a lesser degree.

What is the internal sphincter?

Wesson described the muscle about the internal urethral orifice as consisting of two bundles: an internal U-shaped bundle which surrounds the urethra posteriorly and laterally, but is deficient anteriorly; and an external U-shaped bundle which lies in front of the urethral orifice and embraces the internal bundle lateral to the orifice, to become continuous with the external longitudinal muscle fibers on the posterior aspect of the bladder. Looney and I found, however, that the internal sphincter consists of two portions, namely: an inner and an outer. We have been able to show that the most internal fibers

of the inner bundle form a complete sphincter around the internal orifice of the urethra. The outer portion of the inner bundle appears to loop partially around the urethral orifice and then becomes continuous with the circular fibers of the bladder. These fibers were, in a majority of specimens examined, more highly developed posterior to the urethral orifice than elsewhere. The outer portion of the sphincter lies external to and somewhat below the inner portion. These fibers begin on the posterior wall of the bladder, where they are continuous with its external longitudinal fibers and pass on each side of the ureteral orifice to its anterior surface, where many of them seem to loop around to the opposite side, while others are continued into the external longitudinal fibers of the anterior wall of the bladder which pass into the pubovesical ligaments. In some of our specimens we were able to demonstrate a definite decussation of some of the fibers of the outer bundle across the median plane, above and behind the urethral orifice. The external portion of the internal sphincter extends much farther downward on the urethra than does the internal portion. In fact, it can be traced to the upper layer of the urogenital diaphragm, while the internal portion does not reach to nearly so low a level.

I believe Wesson describes the finding in embryos before the sphincter was fully developed and his findings suggest that the muscle fibers have two points of origin. I do not believe there is a specialized bundle of fibers which form a sphincter. In cases of so-called median lobe enlargement, a group of fibers may be separated and may give the appearance of a separate and distinct structure, but that is an artefact. If the submucous fibers are cut, the deeper fibers often take over their function of closing the internal orifice of the bladder. When fibrosis occurs, especially in early adenomatous hypertrophy, it causes increased rigidity and causes first hypertrophy of the muscle fibers, but if the adenomatous mass pushes up into the bladder, it usually leads to muscular atrophy. In patients with a tabetic bladder, the internal urethral orifice is funnel-shaped unless adenomatous hypertrophy has already developed and in that case we see the usual obstructive lesion. This is very important because resection may not give any relief, not because of the obstruction, but because the bladder muscles are not capable of contracting.

Although it will be more fully discussed in a later paper, I wish to state that I believe there are no special nerves to the internal sphincter. The parasympathetic (sacral) nerves supply the entire bladder except the trigon, and stimulation causes a wave of contraction which spreads over the bladder and down the urethra similar to the contraction of the heart. This contraction wave, together with contraction of the trigon muscles, produces emptying of the bladder. In a few words, the so-called internal sphincter of the bladder consists of a continuation of the bladder muscle fibers which encircle the urethra, and because of the angle formed between the urethra and bladder they are able to compress the internal orifice by tonic contraction.

We now come to the discussion of what we are attempting to do when we do a prostatic resection. When I stated in a previous paper that one of the aims was to destroy the internal sphincter, it was seriously questioned. Subsequent observation has only strengthened this belief. Every resection of the floor of the urethra destroys the trigon muscle. When this is done the internal urethral orifice must be opened by increased intravesical pressure unless the internal sphincter is greatly weakened. In many cases of prostatic hypertrophies the internal sphincter is already destroyed. Often the trigon is unable to open the internal orifice because of the hypertrophy. These patients tell you they cannot void if they attempt to strain. The urologist's problem is to balance the action of the two muscles. If the trigon is destroyed, the sphincter must be weakened or destroyed. When the trigon, by fibrotic repair, is able to resume its function, the ideal result would be recovery of the function of the internal sphincter. Postoperative cysto-urethrograms show plainly that the resection transforms the deep urethra into a funnel with the apex at the verumontanum and the base at the urethrovesical juncture. The frequent statement of patients that after resection they find they must void promptly when the impulse is felt means that the entire load is thrown on the external sphincter, and until it adjusts itself to this, partial incontinence is experienced. I believe many patients regain a part of the sphincteric action of the internal sphincter. I base this on the fact that some patients find, after several months, that seminal fluid is ejaculated at intercourse rather than escaping back into the bladder. It is not sufficient to remove a few muscle fibers, but enough tissue must be removed to leave a wide open sphincter and prostatic urethra down to the external sphincter.

We know that in women, the deeper muscles

may take over the sphincteric action after injury to the submucous fibers. If the semi-elastic tissue about the internal orifice is removed, the deeper muscles may be capable often of closing the internal urethral orifice. Every effort should be made to prevent postoperative fibrosis in this area. I feel that the coagulating current should be used only when hemorrhage cannot be otherwise controlled.

I want to say a few words regarding the resection of infected prostates. I tried it in a few cases and soon realized my mistake. I had hoped, as others claimed, that resection would remove the infected glands, but if one will note the position of the prostatic glands, it is plain that resection would remove only a few small suburethral glands.

Some urologists argue that no harm comes from trying resection in these cases. It has been my fortune, or, you may say misfortune, to care for several such cases and I know of no class of patients who are more uncomfortable. One patient whom I resected seven years ago states that each intercourse is ended with a severe cutting pain which occurs at the time of ejaculation. This pain may persist as pain in the perineum for several days. Others have reported similar experiments. Apparently, adhesions are produced about the orifices of the ejaculatory or prostatic ducts. I have not seen similar symptoms in cases of senile hypertrophy of the prostate. This suggests to me that it is the prostatic ducts, because in hypertrophy I suspect they are usually closed already. More work must be done on the question of why a prostate showing adenomatous hypertrophy may be resected without symptoms such as are seen in a younger man with an inflamed prostate.

It is very difficult to know when to resect these patients. Some have claimed we should resect before the adenomatous change appears. I have always felt that if prostatic massage will yield prostatic secretion or if the urinary symptoms are benefited by the massage, we should delay resection. If the patient has a persistent residual urine, or if we see a large bas fond when we make a cystoscopic examination, resection is indicated. But I do not believe the size of the prostate by rectum, especially in a man under fifty-five, should be an indication for any surgery until a thorough course of prostatic massage is given. If secretion is expressed on massage, I do not believe that the prostate will show adenomatous change. We should resect as soon as we are convinced that there is adenomatous change or when we have a so-called "prostatic bar." If we allow the hypertrophy to proceed too far, the rigidity of the prostate may even involve the region of the external sphincter. The postoperative results are always better in the earlier case, but this is not true if too early.

Fig. 9 will show three different positions in which a prostatic abscess may occur. The abscess of the anterior lobe would drain after simple transurethral incision, but the other two can be drained adequately only by dependent perineal drainage.

CONCLUSIONS

(1) The prostate is not an anatomical entity, although it has a specialized physiological function. It is merely the glandular area of the urethra between the internal urethral orifice and external sphincter.

(2) The impression of division of the prostate into lobes arises from the compression caused by the action of Bell's muscle.

(3) The internal urethral sphincter is comprised of those muscle fibers of the bladder wall which, because of their location, are able to compress the internal urethral orifice.

(4) Prostatic resection should remove sufficient tissue temporarily to destroy the action of the internal urethral sphincter, and to form a funnel whose apex is at the verumontanum.

(5) Prostatic resection does not remove the infected glands in cases of prostatitis.

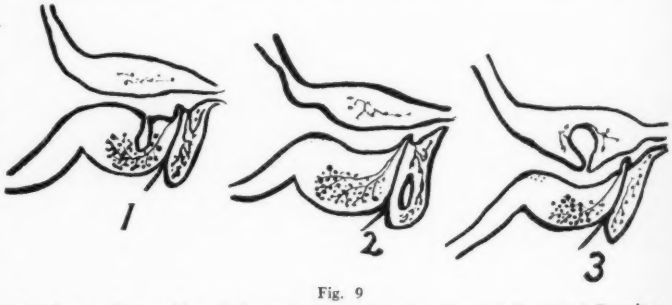

Fig. 9
Drawings to show position of abscess in three autopsy specimens: 1. Lateral; 2. Posterior; 3. Anterior lobe.

DISCUSSION (*Abstract*)

Dr. Miley B. Wesson, San Francisco, Calif.—Dr. Van Duzen is to be commended on the work he has done with Dr. Looney during the past few years on the anatomy of the trigon and bladder neck. His studies have all been made on dissecting room material and his conclusions are based wholly on gross dissections. Consequently his report of finding a circular internal sphincter cannot be accepted until he proves it by means of serial sections prepared with differential stains.

Dragonas, now of Athens, reported from Legueu's Clinic about three years ago a detailed study of the trigon and sphincters of the bladder based upon gross dissections of anatomical material. He stated that there was no real connection between the bladder and the prostate; the trigon was made up of two distinct parts of separate origin, an anterior third and a posterior two-thirds, was not a separate muscle superimposed on the bladder floor, had nothing to do with the instigation of urination, and furthermore, that it did not pass into the urethra. The internal sphincter was an oblique loop proximal to the vesico-urethral juncture and should be considered as merely a thickening of the circular layer of the urethra. He did not believe that the heavy anterior arc from the external longitudinal layer played any part in the sphincteric action. He enunciated a theory of micturition based on these dissections and his knowledge of physics. What he needed was not a series of diagrams of strain and stress, but some photomicrographs from serial sections through the neck of the bladder. Dr. Van Duzen has attacked the problem in exactly the same way as did Dragonas and has demonstrated that all of the conclusions of the latter are incorrect in that they were based on inaccurate dissections and faulty logic.

Fourteen years ago I reported that there was an "anatomical" external sphincter made up of striated muscle and a "surgical" internal sphincter made up of an anterior loop from the longitudinal layer of the bladder and a posterior loop from the internal circular layer. The latter was oblique, one end being in the posterior vesical lip and the other in the roof of the urethra above the verumontanum. A vertical section through the urethrovesical orifice shows two half loops interlocking with a tonsillotome effect. The action is that of a sphincter, but since it is not a muscle whose origin and insertion is in itself, it is a surgical sphincter and not an anatomical sphincter. This study was based on serial sections, both transverse and sagittal, prepared with differential stains of the pelvic organs of many embryos, infants and adults. The drawings were made on glass plates by means of an Edinger projection apparatus and Higgins inks (ten colors). The glass plates were stacked and by means of bright lights the three-dimension model could then be studied. All of these sections were then given to the Carnegie Institute of Embryology, Baltimore, and may be borrowed by anyone who wishes to use them.

Dr. Van Duzen has demonstrated very clearly that the prostate is the neck of the bladder and is made up of muscles which have been invaded by prostate tubules; also that the so-called surgical capsule of the prostate is merely the compressed prostatic tissue that remains after the hypertrophied adenomas have been removed.

Dr. W. W. Looney, Dallas, Tex.—After listening to the various discussions of Dr. Van Duzen's paper, I am convinced that the different men who have entered into the discussion are not necessarily disagreeing with one another as to the anatomy of the prostate as much as their remarks would at first indicate, but that each one is describing a different prostate. It should be remembered that prostates differ in structure with age, and also with the amount of pathologic deviation that may have resulted from previous disease. The prostates that most of you have occasion to examine are not what textbooks describe as normal glands. I note particularly that you are not in agreement concerning the capsule of the gland. Gray's "Anatomy" describes a definite fibrous capsule of the gland. Cunningham's "Anatomy" states that the outer layer of the prostate is composed of interlacing smooth muscle fibers and connective tissue, forming a kind of capsule for the underlying structures. After examining many prostates in the dissecting room, I am forced to agree with Dr. Van Duzen that the normal prostate does not possess a definite fibrous capsule and that its muscle fibers are directly continuous with those of the bladder. In hypertrophied prostates, however, the outer muscle fibers become so dense and fibrous in structure as to make it appear that the gland has a definite capsule. I believe that it has been in such glands that Dr. Davis has observed a capsule.

Surrounding the prostate, but not closely attached to it except in front, is the visceral layer of endopelvic fascia described as the sheath of the prostate. The sheath is continued upward on to the bladder. Between the sheath and the gland is the pudendal plexus of veins (prostatic plexus). These are the structures that give much troublesome hemorrhage in the operation for complete enucleation of the gland.

I wish time would permit further discussion of the sphincter muscle of the bladder just discussed by Dr. Wesson; but suffice it to say, we have specimens of this structure in our laboratory of anatomy that show a definite and complete sphincter muscle immediately surrounding the internal urethral orifice.

PROLONGED INTENSE HEAT IN THE TREATMENT OF PELVIC INFECTIONS*

By GEORGE A. WILLIAMS, M.D.,
Atlanta, Georgia

External heat is time-honored as a valuable agent in the treatment of inflammation. Its popularity is directly proportional to the efficiency and facility with which it may be applied to the tissues affected. No ophthalmologist would fail to include it in his armamentarium, nor would the surgeon who deals with inflammations of the extremities. There are few gynecologists at the present time who do not

*Read in Clinical Session, Obstetrics and Gynecology, Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

make some use of heat in the treatment of pelvic inflammatory diseases in spite of the more complicated methods required for its application in this field.

Crossen[1] and Simpson,[2] in 1909, called attention to the importance of conservatism in reducing the high mortality of the operative treatment of acute salpingitis, which at that time was regarded as an urgent surgical emergency. Since then the use of conservative measures in the treatment of pelvic inflammation has steadily grown in favor, both as a substitute for and as an adjunct to operative treatment.

The vaginal douche is an age-old method of treatment of pelvic disease. Mentioned by Hippocrates, who used it to wash away the discharges so all-important to his humoral system of pathology, the douche is even now too often used in a similar manner, or merely as a vehicle for the administration of frequently impotent drugs. The truth is that the conventional douche consists merely of the act of flushing the vagina, and as such is a very inefficient means of administering heat. Even in the prone position little more than one ounce of water may be introduced into the vagina without spilling, while with the apparatus to be described the author has found that the average vagina has a capacity of eight ounces when distended with a pressure of one pound. The vaginal capacity varies little with size, marital state, or parity, but it is strikingly small in the infantile state (*Table 1*).

Table 1

Height	Weight	State	Parity	Capacity c. c.
64	116	M	1	270
62	114	M	1	270
63	110	M	1	265
61	102	M	1	260
63	118	M	1	255
62	118	M	1	255
61	100	S	0	250
64	136	S	0	250
61	150	M	2	250
70	190	M	4	250
64	122	S	0	245
63	138	M	†	245
62	118	M	1	240
60	128	M	0	240
62	112	M	1	240
62	150	M	†	215
68	138	M	†	185
60	100	M	†	180
62	142	M	†	95
*64	239	M	†	68

*Extreme virilism.
†Sterility other than inflammatory.

In 1923 Gellhorn[3] discovered that the vaginal and cervical epithelium would tolerate higher temperatures (120° F.) than the vulvar skin. He devised the prolonged hot douche to be taken in the dorsal position with the pelvis elevated and the skin to be protected with petroleum jelly. An extremely important step in pelvic physiotherapy, the prolonged hot douche, remains our most valuable agent in the home treatment of pelvic inflammatory diseases.

Many women either cannot, or will not avail themselves of the prolonged hot douche, however. Seeking other measures, Cherry[4] and Gellhorn[5] have reported excellent results in the treatment of pelvic inflammation with diathermy. Other authors have used diathermy with varying success. Requiring heavy, expensive apparatus and a certain degree of special training in its use, diathermy and its ever-present *bete noir,* the burn, has never achieved general use.

Of all methods of applying heat to the female pelvis, that devised by Dr. C. R. Elliott[6] and known by his name is probably the most efficient. Thermostatically controlled hot water is circulated by electric pumps, though a specially constructed distensible rubber bag which encircles the cervix uteri and lifts the uterus and adnexa from the lower pelvis. The pressure for distention of the vagina is mechanically controlled and may reach two pounds. Temperatures as high as 130° F. are gradually reached and maintained for an average time of one hour. The vulva is protected by insulating with wet gauze. By means of this method the temperature of the adjacent tissues is elevated, reaching as high as 104° F. in the urethra and 106° F. in the rectum. The immediate effects are striking. An intense hyperemia of the parts occurs and a copious outflow of the cervical mucus results. The cervix is softened and relaxed. There is usually an immediate relief of pain, lasting for several hours. Repeated applications result in rapid absorption of exudates and resolution of adhesions. Even in a "frozen pelvis" the uterus usually becomes well mobilized. In those cases which eventually require operation, the striking freedom from adhesions permits conservation of tissues which otherwise could not be saved.

The results of such treatment upon the circulation must be striking. In 87 per cent of one series of cases the average increase in leukocytes was 17 per cent. The local increase in circulation must be enormous, for the author has found that in some cases eight ounces of water introduced into the vagina at 130° F. are reduced in

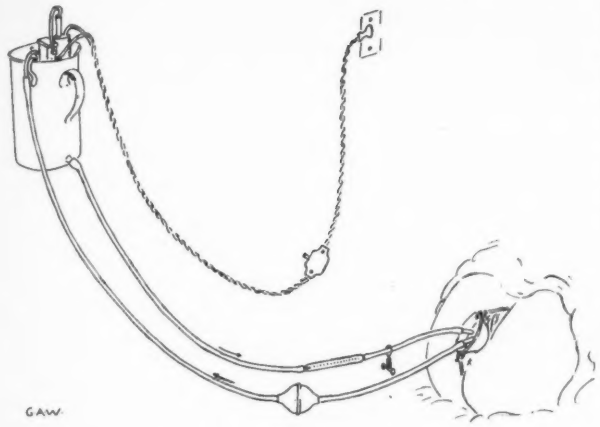

Fig. 1

The author's apparatus assembled for general use. The thermoregulator is set at 130° F. and lower temperatures must be secured by observing thermometer and using switch in the electric line.

temperature to 124° F. at the end of thirty seconds. This means that 3,000 calories per minute have been conveyed away by the circulation.

The Elliott treatment has attracted universally favorable attention. A few burns, usually inconsequential, have been reported. These are thought to be due to technical errors and not primarily attributable to the method. The only obstacle to the general use of the Elliott method is the complex and expensive apparatus required. At the present time its use is largely confined to the better equipped hospitals, while the bulk of pelvic inflammatory disease remains to be treated by the general practitioner who is already overburdened by equipment and operating expense.

The author has assembled an apparatus from standard surgical and laboratory supplies which has accomplished satisfactory results. It differs from Dr. Elliott's apparatus in lacking mechanical control of circulation and pressure, thus eliminating the most costly items in his machine.

Water, heated by a thermoregulated 300 watt immersion heater in a two-quart irrigating can, flows into a hard rubber nozzle, to which is attached a male contraceptive sheath as an intravaginal bag. An infusion thermometer and a stop-cock are placed in the line between the reservoir and the nozzle. Pressure of the distending fluid is produced and maintained by the height of the water column, never exceeding one pound (27½ inches). The water is returned to the reservoir by means of a rubber bulb hand pump in the outflow line. Glass valves are inserted above and below the pump to direct the flow constantly toward the reservoir, where the water is reheated. A snap switch is placed in the electric cord to the immersion heater to provide instant control.

The technic of application is simple. The sheath is attached to the nozzle in the steps indicated in the accompanying diagram. The nozzle, properly lubricated, is inserted in the vagina and fixed to a sanitary belt. It is important that the labia not be compressed between the nozzle and the pubic rami, the ischemia so produced inviting burns in spite of the insulation of the hard rubber nozzle. The initial water temperature should not exceed 120° F. and is tested by opening the stop-cock and allowing the water to run past the thermometer until the

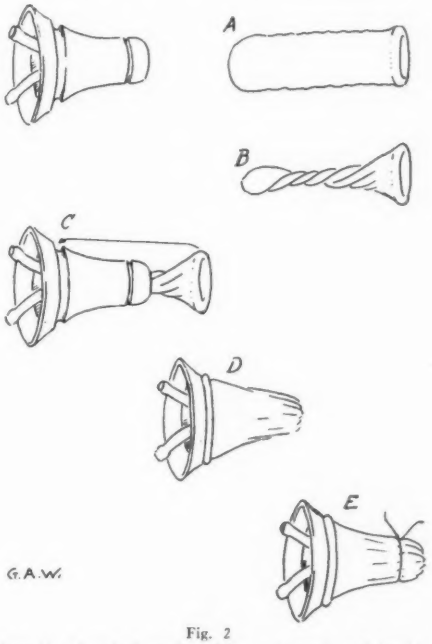

Fig. 2

Assembly of vaginal nozzle and bag. A, nozzle and sheath. The flared end of the nozzle is perforated for attachment of tapes. B, C, D and E, steps in fitting and fastening sheath to nozzle. The nozzle is then lubricated with a jelly, inserted in the introitus, and attached by tapes to the patient's sanitary belt.

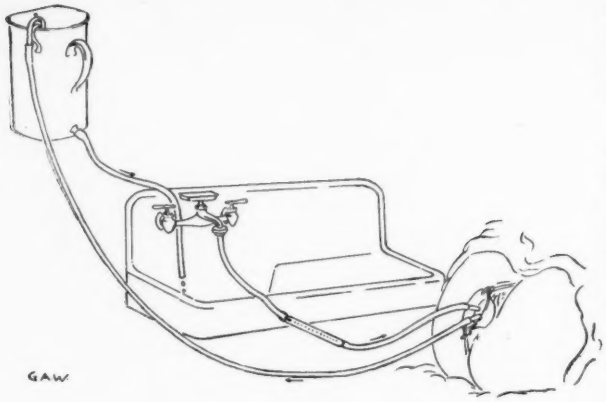

Fig. 3

Simpler and less expensive apparatus for use where suitable hot and cold water supply is available. The thermometer must be watched closely for fluctuations in water temperature.

reading remains constant. Both inlet and outlet tubes are then attached to the nozzle and the stop-cock is opened, allowing the bag to fill. Distention of the vagina should not be too rapid, for this will produce pain. Initial pressure should be low, to be increased as the patient's improvement permits. Pumping is then begun and the temperature allowed slowly to reach 125° F. for the initial treatment and as high as 130° F. during subsequent treatments. The duration of treatments should vary from thirty minutes to one hour, depending upon the patient's reaction. Caution must be exercised in the treatment of hypotensive and debilitated patients, for the systemic effect is that of a hot bath and may be very innervating. To terminate the treatment the stop-cock is closed and the bag is deflated with the pump. Careful inspection should be made to detect possible burns at the end of each treatment.

When the physician's office or the patient's home is equipped with a suitable hot water supply, the apparatus can be even more simple. The thermoregulated immersion heater and the bulb pump may be dispensed with. Water from the supply pipes at proper temperature is carried directly into the inlet tube. The thermometer in this case should be close to the faucets so that proper mixing of the hot and cold water may be carried out. The pressure of the distending fluid is obtained by having the outlet tube empty into the irrigating can at the proper height, whence it flows back to the lavatory drain. The expense of the apparatus is negligible.

It is not within the scope of this article to discuss the indications for the use of heat in gynecologic conditions. Attention is called, however, to the heat treatment of two frequently intractable conditions, acute gonorrhea and *Trichomonas vaginalis* vaginitis. Clinical and bacteriological cure of uncomplicated gonorrheal urethritis and cervicitis has been accomplished in four cases in five to six weeks. Daily treatments with heat are given. The appearance of an angry cervix is changed to one of normal appearance in three or four days in some cases.

Fig. 4

Roentgenogram of pelvis showing bag distended with air under one-pound pressure.

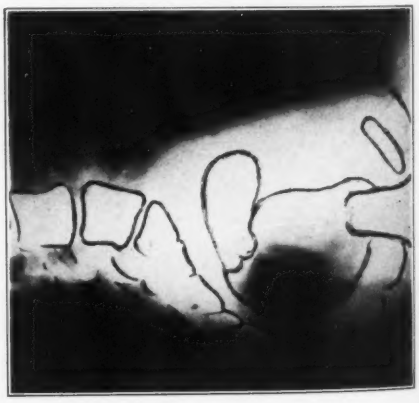

Fig. 5

Lateral view of pelvis showing air distended bag in position. On account of hazy detail of soft tissues, the principal landmarks have been outlined by tracing.

At the end of four weeks the cervix and paraurethral glands are cauterized.

In *Trichomonas vaginalis* vaginitis heat treatments are given daily and a douche of sodium perborate 1:1000 is to be used by the patient each night. Four cases have all presented clinical and bacteriologic cures at the end of a month's treatment. One of these had resisted other methods for three months. There have been no recurrences. While this is a very small series, the results are in marked contrast with those obtained by other means.

In conclusion, the purpose of this presentation is only to encourage a more widespread application of the principles outlined by Gellhorn, Cherry and Elliott through the use of simple and inexpensive but efficient apparatus.

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TRICHOMONAS VAGINALIS AND MONILIA ALBICANS AS CAUSES OF LEUKORRHEA*

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There are many causes of leukorrhea, but I should like to speak of two very common causes of a discharge and a vaginitis which have been overlooked by many physicians, and these two are: *Trichomonas vaginalis*, a protozoan, and *Monilia albicans*, a fungus. They are overlooked because a fresh smear is seldom examined in cases complaining of a discharge. Also, few technicians are told to look for these organisms and few are able to recognize them.

This is a report of an examination of over 3,200 women, pregnant and non-pregnant, over a period of three years for *Trichomonas vaginalis* and *Monilia albicans* and also for other causes of their discharge. This report covers the treatment of many of these women.

SIGNS AND SYMPTOMS

The signs and symptoms of these infections are more or less characteristic. The discharge may be similar in both infections. In cases with Monilia, the discharge is usually thin and watery, but a characteristic thick, cheesy, greenish material may be seen here and there on the vaginal walls. In cases with Trichomonas the discharge is usually whitish-yellow in color and collects in the posterior fornix of the vagina and between the labia.

The odor is usually more offensive in cases infected with Trichomonas; the discharge of Monilia is less likely to have so foul an odor. The most malodorous discharges that I have ever seen were in cases infected with *Trichomonas vaginalis*, this infection being at times second to carcinoma of the cervix.

Vaginal Lesions.—In many cases of *Trichomonas vaginalis* there is the so-called "scarlet fever" or "strawberry" vagina, which is pathognomonic. On the other hand, with Monilia infection there are usually the white flakes or small raised white spots lying here and there on the vaginal walls. These small white spots, which are about the size of a match head, are seen more commonly in gynecological patients while large irregular patches are common in pregnant patients. There is usually an area of hyperemia under these flakes or spots. The flakes are easily swabbed off (*Fig. 1*).

In both infections there may be an acute vaginitis. The vaginitis in many cases of *Monilia albicans* is a subacute rather than an acute vaginitis. It is mild. *Monilia albicans* produces many cases of acute vaginitis in pregnant women. Trichomonas is capable of causing an acute vaginitis and it is one of the common causes of a vaginitis in pregnant as well as in non-pregnant women. Where there is an acute vaginitis produced by any one of these infections, the signs and symptoms are the same.

The most pathognomonic complaint that patients give when they have Trichomonas is that they have used all sorts of douches and have been treated by several or many physicians for their discharge, yet the discharge still persists. Trichomonas may be suspected also when a patient complains of a discharge which causes an itching and scalding of the external genitalia,

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and when she complains of bleeding after intercourse and no abnormality is noted except the "strawberry-like" spots. Trichomonas is to be suspected in every case of leukorrhea and should be ruled out before treatment is instituted.

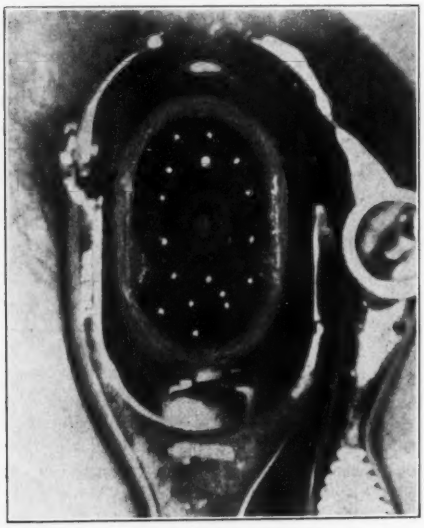

Fig. 1
Monilia albicans during the gynecological period. Note the round white spots. These are irregular flakes during pregnancy.

Fig. 2
Monilia albicans on plate.

Monilia albicans is to be suspected: (1) when a pregnant patient complains of a discharge; (2) when a woman with diabetes has a discharge; (3) when a patient has glycosuria and a discharge; (4) when there is merely a discharge that soils the under clothing; (5) when there is an itching of the external genitalia, which is worse at night. *Monilia albicans* should be ruled out in every case of leukorrhea in pregnancy. It is a very common cause of leukorrhea and vaginitis in pregnancy.

For the sake of clarity I will give a table of the differential diagnosis of *Trichomonas vaginalis* and *Monilia albicans:*

TRICHOMONAS VAGINALIS	MONILIA ALBICANS
(1) Discharge increases after the menses.	(1) Discharge increases before the menses.
(2) Persists despite most methods of treatment.	(2) Easily treated.
(3) Constant leukorrhea in most cases.	(3) Remission in the production of a discharge.
(4) Itching around the vulva common.	(4) Itching around the vulva not so common.
(5) Discharge scalds or burns the thighs wherever it comes in contact with them.	(5) Discharge apparently does not burn or scald thighs.
(6) Local application of gentian violet alone will not clear the infection.	(6) Gentian violet clears infection almost instantaneously.
(7) Seen in pregnant as well as non-pregnant.	(7) Seen most commonly during pregnancy; not so common in gynecological patients.
(8) (a) Diagnosed by examining a fresh smear. (b) Not so often seen in stained smears.	(8) (a) Diagnosed by a fresh smear. (b) Seen best in stained smears.
(9) Produces a malodorous discharge.	(9) Apparently does not produce a malodorous discharge.
(10) A protozoan.	(10) A fungus.
(11) Causes itching right after menses, and itches more often in day time.	(11) Causes itching most any time and more so at night.

DIAGNOSIS

All that I can add to this part of the paper other than that which has been given by me in previous reports is that one should obtain an adequate amount of discharge for a thick smear examination and should classify the cases accordingly. From this the severity of the infection can be estimated in many cases. In examining the fresh smears, I tried to obtain the same amount of discharge; to use the same amount of tap water every time; to spread the same amount of this mixture on the slide in order to find whether there was any relation be-

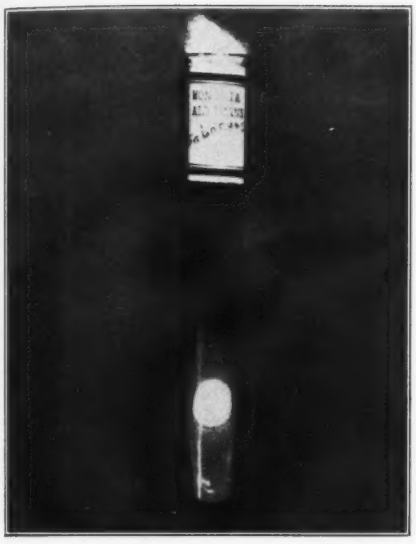

Fig. 3

Monilia albicans on Sabourraud's slant. When these cultures were inoculated, they produced typical Monilia albicans infection of the vagina and in some cases itching of the vulva.

tween the number of Trichomonads, bacteria, degenerated epithelial cells, and so on, and the signs and symptoms found. In doing so, I used the following terms:

1-plus, a trichomonad seen now and then with almost all the epithelial cells normal, but always some degenerated cells. Seldom pus cells or red blood cells. Highly acid in reaction. pH. 4.4 4.6.

2-plus, two to three Trichomonads in the field with about half of the epithelial cells degenerated, a few pus cells, few red blood cells. Slightly acid in reaction. pH. 4.6-4.9.

3-plus, three to five Trichomonads in a field, about three-fourths of the epithelial cells degenerated. Pus cells. Acid in reaction. Red blood cells. Few bacteria. Slight odor. pH. 4.9-5.7.

4-plus, the field literally covered with Trichomonads. Many pus cells. Many bacteria or few. Very bad odor. The field is merely a mass of degenerated epithelial cells, pus cells, red blood cells or no red blood cells, all sorts of tissue debris, and Trichomonads. pH. 5.7-7.6.

Patients in group 3-plus and 4-plus all had a discharge with burning or itching or a vaginitis. Those in 2-plus could control the discharge, itching and burning by constant douching. They seldom suffered from itching and burning. In the 1-plus group there were no symptoms, but there was always an abnormal discharge.

Trichomonas vaginalis can also be diagnosed by stained carbol fuchsin smears, but after Miss Lilia Behrns, a Jefferson Davis Hospital technician, and I examined about 500 stained smears, we found that stained smears were not so accurate as the fresh smears. In a stained smear one may see the organisms as in *Fig. 5-B*, and sometimes one sees just the brown flagella lying scattered over the entire field as in lower right hand corner of the figure. This is also indicative of *Trichomonas vaginalis* when found.

TREATMENT

The treatment of Trichomonas is not so simple as one would think when he sees how easy it is to destroy the organism under the microscope.

I have been treating my cases according to the laboratory findings: 1-plus, 2-plus, 3-plus, or 4-plus. In 1- and 2-plus cases, I give the following douches, as described in the State Journal and elsewhere:[1] bichloride of mercury, sodium perborate, Lugol's solution; and in 3- and 4-plus cases I have been using the following method, with good results. (I have tried kaolin, starch and soda, alone and in various proportions, and found that corn starch alone gives me the best results.) It is also more comfortable to the patients.

The vagina is cleaned out with tincture of green soap solution, a one-ounce male asepto syringe (end slightly enlarged by grinding on an

Fig. 4

Monilia albicans as seen in a fresh and in a stained smear. Monilia albicans can be diagnosed by a fresh smear as well as by a stained one (in my experience).

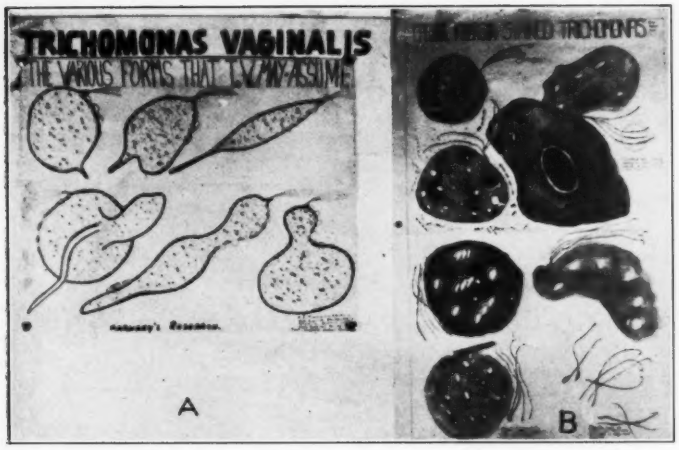

Fig. 5

Shows on the left (A) the various forms that *Trichomonas vaginalis* may assume and on the right (B) the appearance of carbol fuchsin stained *Trichomonas vaginalis*. Note the free flagella lying in the lower right hand corner. Sometimes this may be all that one sees in a stained smear and this is diagnostic of *Trichomonas vaginalis*.

emory wheel) full of sifted corn starch is inserted firmly at the introitus of the vagina, and by quickly pressing the bulb of the syringe the entire vagina can be stretched and its walls covered with a thick layer of the corn starch (remembering that drying of the vagina is what we want). The patient is instructed to leave the corn starch in the vagina for about two days and then on the evening of the second day to wash out the starch with warm tap water and return to the physician's office. On this day the vagina is inspected and is blown again with corn starch and left in for two more days. This is usually done three times; in some cases one will have to treat the vagina four to five times.

After the powder treatment, the patient is given U.S.P. lactic acid douches, teaspoonful to a quart of water, once a day. This is given in order to restore the normal bacteria flora. One can also order glucose (white karo) douches, one tablespoonful of this to a pint or quart of warm water, the sugar being broken down into lactic acid in the vagina by the Döderlein bacilli.

The patient is instructed to take douches, any of the above, three times a day during her next three menstrual periods, and twice a day if she works away from home. She is to report back to the office after her first menstrual period for another course of powder treatment. The treatment will clear up most of the cases, but if she still has infection after this has been tried, then the scrubbing, painting and packing method is used. In a follow-up of 223 cases treated by the scrubbing method, of which some had bladder irrigations and a silver protein compound (neosilvol) instilled into the rectum, 96 per cent were cured over one year's time. If the case is not cured after the latter has been tried, then the bladder is irrigated with boric acid solution and argyrol instilled. The anal hygiene is gone into and finally the prostate and urethra of the husband is treated.

The only addition that I have to the treatment of *Monilia albicans* is to use 2 per cent gentian violet in 75 to 90 per cent ethyl alcohol or 5 to 7 per cent aqueous gentian violet instead of the weaker solutions given in my previous

Fig. 6

Oil emmersion carbol fuchsin stained *Trichomonas vaginalis*. Note the vacuoles and flagella.

Fig. 7

Carbol fuchsin stained *Trichomonas vaginalis*. This shows how well *Trichomonas vaginalis* can change its shape. Note the axostyle (tail) and flagella are now almost at one end while normally they are at opposite ends. Note vacuole and part of epithelial cell.

reports. These solutions are more penetrating and will destroy the infection more quickly.

Many cases of Monilia will disappear after delivery. This has been substantiated by clinical evidence. Fifteen per cent of 500 pregnant patients had Monilia, while after delivery only 9 per cent had *Monilia albicans*. For a complete report on *Monilia albicans* in the vagina, see Plass.[2]

LIFE HISTORY AND MODES OF TRANSMISSION

Little is known about the life history of *Trichomonas vaginalis, buccalis* and *intestinalis*. We all know that if the life history of a disease-producing organism is known, then we can interrupt this chain and in many instances cure the infection.

The life history of the Trichomonads has been of interest to me, especially the *Trichomonas vaginalis*.

Is the organism transmitted as *Trichomonas vaginalis?* Infection may be acquired by: vulval intercourse; using a recently infected douche syringe; using an infected towel, if it is kept warm and damp; from fresh discharge on a toilet seat; using a recently infected bathtub, and by sexual intercourse.

Here I should like to report a case that could have been and might have been a real source of infection by sexual intercourse. This was a negro man 23 years of age. He had intercourse and two days later he was sent to the penitentiary for a year because of burglary. He states that he has had a peculiar itching just inside of the meatus and on the head of his penis for the past year. He has not had intercourse since the time given above. He still has the itching of his penis. Urine and fresh smear examination revealed many Trichomonads of the vaginal type, pus and urethral epithelial cells, a typical smear for a case of urethritis. This patient has an urethritis apparently due to *Trichomonas vaginalis*.

If only a few Trichomonads reach the vulva, here they may grow and multiply and swim back into the vagina. The infection is more likely to take place if the woman is menstruating. In all cases in which exposure to Trichomonas occurred during the menses acquired the disease, while some of the cases in which inoculation failed were exposed between the menses. In most cases *Trichomonas intestinalis*, few in number, are apparently living between the labia until the woman menstruates; then multiplication takes place and the Trichomonads swim back into the vagina in the menstrual fluid. I have found *Trichomonas intestinalis* between the vulva.

I also believe the infection is obtained from the mouth by the many ways that one may think of. (See Curtis, "Text of Gynecology and Obstetrics," 3:429 and 432, for further data of mine.)

I have seen *buccalis*-like forms in fresh vaginal smears many times. They were very much or exactly like the buccal forms, in size, habit, and motion. I have also seen *intestinalis*-like forms in fresh vaginal smears. They were very much or exactly like the intestinal forms, in size, habit, and motion. In some smears I have seen true *vaginalis, buccalis* and *intestinalis* forms in fresh smears and the patients apparently had their source of infection both from the mouth and rectum. (See Davis, "Gynecology and Obstetrics," chapter 17, vol. 2, page 19.)

Every case of pyorrhea alveolaris that I examined had *Trichomonas buccalis*. Many of my positive cases here at the Jefferson Davis Hospital and the John Sealy Hospital had pyorrhea. Some had *Trichomonas intestinalis*. Many of my cases had *Trichomonas intestinalis, Trichomonas buccalis*, as well as *Trichomonas vaginalis*.

PATHOGENICITY

It is rather widely agreed now that this organism is pathogenic.

Since my last reports I have inoculated more patients with *Monilia albicans* and *Trichomonas vaginalis* and now I can say with more certainty that these organisms are pathogenic. I have produced pathognomonic trichomonal lesions in several patients and I can say that the "strawberry" vagina, as shown in the lantern slides, is pathognomonic of Trichomonas vaginalis and I have not seen it in any other condition.

Although the earlier writers did not believe that Trichomonas were pathogenic, at present almost all writers believe that they are. In Germany, Seitz[3] and Hoehne[4] believe that they increase the virulence of the associated bacteria. Hoehne, of Germany, has also noticed that when the inflammatory phenomena disappeared with the *Trichomonas vaginalis* stained streak preparations showed an unchanged bacterial flora.

I also inoculated bacteria associated with *Trichomonas vaginalis* and these did not produce a discharge, but when *Trichomonas vaginalis* and the associated bacteria were inoculated a leukorrhea and pruritus were complained of, and in some cases an acute or subacute vaginitis was produced. The worst cases of pruritus vulvi that I have ever seen were in patients inoculated with *Trichomonas vaginalis*.

MALE AS SOURCE OF TRICHOMONAS VAGINALIS

Lately, I have been examining prostatic secretions of men for Trichomonads. In the examination of 50 men I failed to find Trichomonads. Yet I believe that in some cases there are Trichomonads in the prostate, because some cases which I cured had recurrences as soon as sexual intercourse was started again. The negro man reported in this paper might have been a real source of spread of the disease.

At exhibits and in the discussions of papers that I have read on *Trichomonas vaginalis*, physicians have told me that they have found Trichomonads in the urine of men and some told me that they have found them in the urine of male children.

Manwell[5] described two cases of Trichomonas in the male. He also reports 33 women who harbored the *Trichomonas vaginalis*, 22 of whom had definite urinary symptoms; 7 of the 20 with urinary symptoms had sensitive urethral meatuses, which were smaller than a 26 F. sound. Dr. E. W. Bertner[5] reports 38 cases of *Trichomonas vaginalis* from January 1, 1934, to September 1, 1934. Three of these had a Neisserian infection; 10 per cent had Trichomonas in the urine. Stuhler[6] reports a case of *Trichomonas vaginalis* in the prostatic secretion. Riba[7] reports 4 cases in prostatic secretions.

TRICHOMONAS VAGINALIS AND POSTOPERATIVE LEUKORRHEA

In a follow-up of 50 postoperative cases, it was of interest to note that 70 per cent of those who complained of leukorrhea had *Trichomonas vaginalis* and were relieved of their discharge when the Trichomonads were destroyed. So, I believe that many of the postoperative cases of leukorrhea are apparently due to *Trichomonas vaginalis*.

In another series of cases, I obtained a smear before the patients were operated upon. In this series, I picked those in 1- and 2-plus classification, with just a few Trichomonads in the field which were not giving the patients much trouble. I did not institute any treatment whatsoever.

When this series came back for a follow-up many of these patients had a profuse yellow-white, malodorous discharge, marked amount of itching of the external genitalia, and the Trichomonads were in the 3- and 4-plus classification. Many of these cases were hard to cure because the Trichomonads had gained a strong foothold.

CONCLUSIONS

(1) *Trichomonas vaginalis* and *Monilia albicans* are common causes of leukorrhea and a vaginitis.

(2) *Trichomonas vaginalis* is capable of causing a malodorous discharge.

(3) Usually in Monilia infections spots are seen in the vaginas of gynecological, and flakes in obstetrical patients.

(4) The differential diagnosis of *Trichomonas vaginalis* and *Monilia albicans* has been described.

(5) A diagnostic classification of *Trichomonas vaginalis* has been given.

(6) The dry method of treatment with corn starch is given.

(7) It is concluded that 2 per cent ethyl alcohol gentian violet or 5 to 7 per cent aqueous gentian violet is better for the treatment of *Monilia albicans*.

(8) A case of *Trichomonas urethritis* has been reported.

(9) The possible modes of infection are discussed.

(10) The male is apparently a source of infection and reinfection.

(11) *Trichomonas vaginalis* is a common cause of postoperative leukorrhea.

(12) *Trichomonas vaginalis* is recognized as a pathogenic organism.

ACKNOWLEDGMENTS

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Thanks are also due to Dr. J. Reese Blundell and Dr. W. E. Bell, urologists, Houston, Texas, for helping me in the examination of prostatic secretions for *Trichomonas vaginalis*. Many thanks are due to the Gynecological and Obstetrical Staff of the Jefferson Davis Hospital. Thanks are due to Dr. J. C. Michael, dermatologist of Houston, Texas, for his aid in culturing *Monilia albicans*.

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Jefferson Davis Hospital

DISCUSSION (*Abstract*)

Dr. Elbert Dunlap, Dallas, Tex.—I am quite familiar with the extensive and careful study, both in clinics and in the laboratory, that the essayist has given to these interesting gynecological conditions. This accurate work has taught many clinicians to master rather easily the technic of finding the micro-organisms and to apply effective, simple treatments which will eradicate them and relieve the symptoms produced by their presence.

In pregnancy the *Trichomonas vaginalis* causes marked vaginal irritation and often is quite annoying. In pregnancy the treatment should be administered cautiously, as very violent efforts at vaginal cleansing followed by the application of strong chemicals may be factors in producing abortion or miscarriage.

THE VALUE OF HYPERPYREXIA IN THE TREATMENT OF BRONCHIAL ASTHMA*

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and
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The production of artificial fever by high frequency currents was first reported by Neymann and Osborne[1] in 1929. King and Cocke[2] and several other workers followed in 1930 with their reports dealing with various methods for the raising of body temperatures. The clinical application of electropyrexia was also introduced, and has been further developed by Neymann and Osborne.[3] All of their work along these lines, however, and as a matter of fact the great part of the literature to date dealing with electropyrexia applies almost specifically to general paresis and diseases of the central nervous system.

In the past four years, however, three groups working independently of each other in the United States and one in Europe began applying this method to the treatment of bronchial asthma. This disease, or rather syndrome, has proven to be very unyielding to therapy. The treatment of bronchial asthma by electropyrexia was first reported by Feinberg, Osborne and Afremow.[4] Seventeen patients suffering from intractable bronchial asthma who had proven to be refractive to conventional treatment were given artificial fever by diathermy. Two treatments comprised a course, but one case received seven. Their theoretical basis for employing this measure, according to their opening paragraph, was the clinical observation of temporary relief from asthmatic attacks associated with febrile infectious diseases, especially if the infection was outside of the respiratory tract. Their biochemical studies consisted of complete blood counts and the acid-base balance. Their results were favorable and encouraging. They[5] made a further report simultaneously with Miller and Piness[6] and Leopold and Stewart.[7] Their final report[8] of thirty-seven cases has since appeared. In this final report their laboratory data included weight, blood pressure, vital capacity, results of urinalysis, complete blood count and the chemical composition of the blood, particularly the acid-base equilibrium, nonprotein nitrogen and

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urea nitrogen before, during and after each treatment. The reasoning of Miller and Piness[6] in employing hyperpyrexia was based upon, first, the fact that asthmatics experienced relief following bouts of fever and, secondly, that parenteral administration of non-specific protein frequently gave periods of relief. They did not report any biochemical studies. Their conclusions were that there was slight improvement. Leopold and Stewart[7] quote Chapman as making the observation one hundred and fifty years ago that asthma was often relieved following active malarial infection. In their report they present charts showing the relief from asthmatic attacks associated with various infectious diseases such as erysipelas and osteomyelitis. They then treated two patients with artificial fever produced by diathermy, giving one a single treatment, the other ten treatments during a period of sixteen days. Their laboratory study was limited to the white blood count. Then there was a period of quiescence. Interest was revived when Feinberg, Osborne and Steinberg[8] and Cameron,[9] of Tumbridge Wells, England, published their reports. The details of Cameron's communication will be considered later.

Neymann,[10] in a review of electropyrexia, states:

"The whole subject of hyperpyrexia is in an almost chaotic experimental stage of development. It is to the physician as an infant industry is to the merchant. The infant is growing and spreading out. With equal certainty all sorts of unfounded, prejudicial and nonsensical claims will be made for and against this type of therapy." And especially referring to the work of Miller and Piness and Leopold and Stewart: "It is evident that some of these workers have not mastered the method of sustaining fever for any appreciable time and these negative results only point the way to a more thorough investigation of the subject."

We, therefore, believe that a report of the therapeutic results obtained in a larger group of patients suffering from bronchial asthma and treated with hyperpyrexia would not be amiss. In order to test the practical clinical value of this therapy we have ignored all allergic manifestations, at the same time attempting a physiologic and biochemic survey to throw more light on the metabolic changes which take place. Therefore, fifty patients suffering from bronchial asthma, regardless of type, were treated with electropyrexia. These patients have now been under observation for periods of from three to eight months.

Our theoretical basis for expecting a marked measure of relief from this type of therapy involves principles in addition to those which were suggested by the above named workers. While we agree with the observations that bouts of fever of infectious origin usually give temporary relief from active bronchospasm, we[11] have pre-

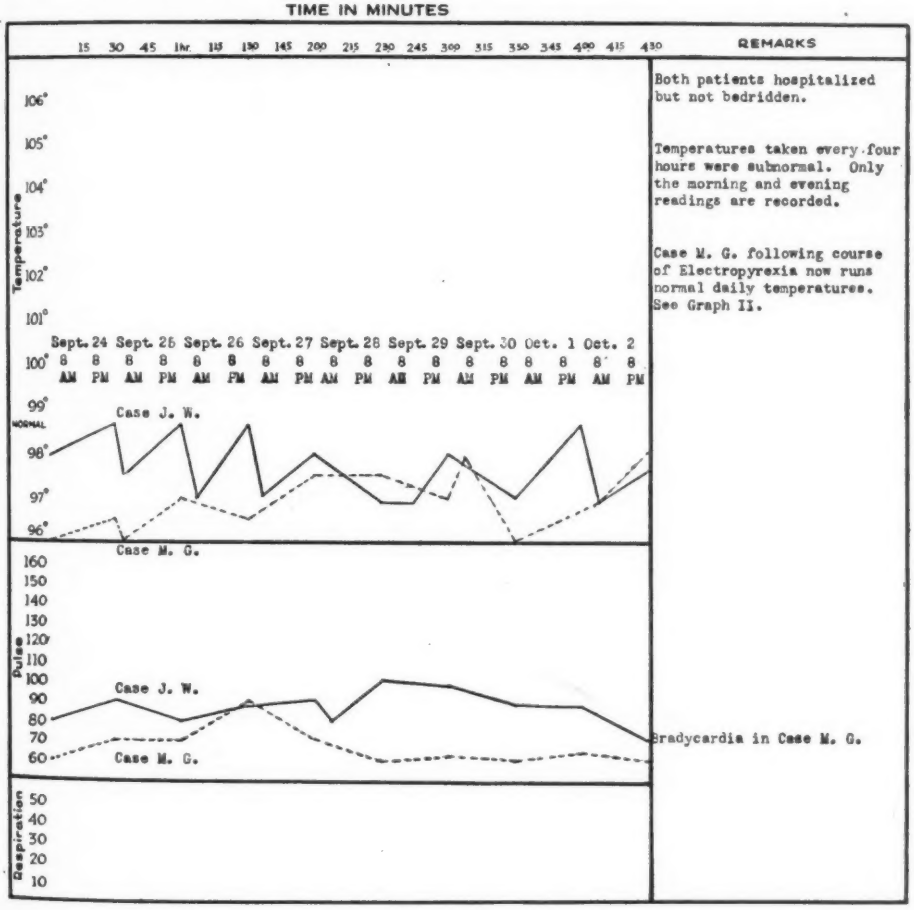
Table 1

ILLUSTRATING THE LOW METABOLIC EBB IN TEN ASTHMATICS MEASURED BY A DECREASE IN NORMAL LEVELS OF VARIOUS PHYSIOLOGICAL CONSTANTS. ALSO SHOWING THE CONSTANCY OF HYPERACIDITY, DECREASE IN UREA AND PRESENCE OF INDICAN IN THE URINE. NOTE THE REMARKABLY CONSTANT FINDING OF A DRY, SCALY AND NON-FUNCTIONAL SKIN

Patient	Avg. Daily Oral Temp. Degrees	B M R	B P	Mg. Blood Sugar	Gas. Acid F.	Gas. Acid Tot.	Deg. Acid	Urine 24 Hours Sp. Gr.	Urine 24 Hours Urea Grams	Urine 24 Hours Ind.	Skin Appearance
H.M.—25—Male	97.6	—12	85/50	87	0	15	90	1026	12	++	Dry, scaly, sallow
P.M.—35—Female	96.8	—10	90/60	90	5	12	65	1015	14	+	Dry, icteric, thick
J.W.—37—Male	96.6	—15	87/40	85	0	10	110	1030	8	++	Dry, eczematoid
M.M.—48—Female	97.8	—12	108/70	87	0	6	102	1020	10	++++	Dry, thickened, scaly
M.G.—54—Female	96	—18	102/70	85	5	15	90	1025	12	++	Dry, sallow, thick
M.B.—29—Female	97	— 6	98/60	92	10	18	62	1024	14	+	Slight moisture
D.B.—7—Male	98	+ 2	90/55	85			102	1018	8	+	Dry, eczematoid
G.S.—8—Female	98	+ 2	90/60	87			64	1010	8	+	Dry, eczematoid
M.N.—10—Male	96.8	—10	85/50	85			72	1008	12	0	Dry, scaly
B.L.—16—Male	97	—12	88/55	87	2	12	80	1016	10	0	Dry, eczematoid

viously called attention to a composite clinical syndrome which we have crudely termed the picture of sympathetic nervous system collapse. These conclusions were based on a study of four hundred cases. In this study we found that the great majority of asthmatics presented a clinical syndrome characterized by a dry, sallow, non-functioning skin, delayed and inefficient kidney action, delayed gastro-intestinal and excretory function, a tendency to subnormal temperature, a low blood pressure, a low basal metabolic rate, hypoglycemia and in some cases even bradycardia. Typical examples are shown in *Table 1.* If this picture as a whole indicates anything it certainly indicates an individual whose general metabolism, circulation and well-being are at a low ebb. Reasoning along these lines, if it were possible to raise the general metabolism and increase the circulation of the organs of secretion, an improvement in the terminal symptoms, namely, bronchospasm, might be expected. Fever of infectious origin might produce precisely such results.

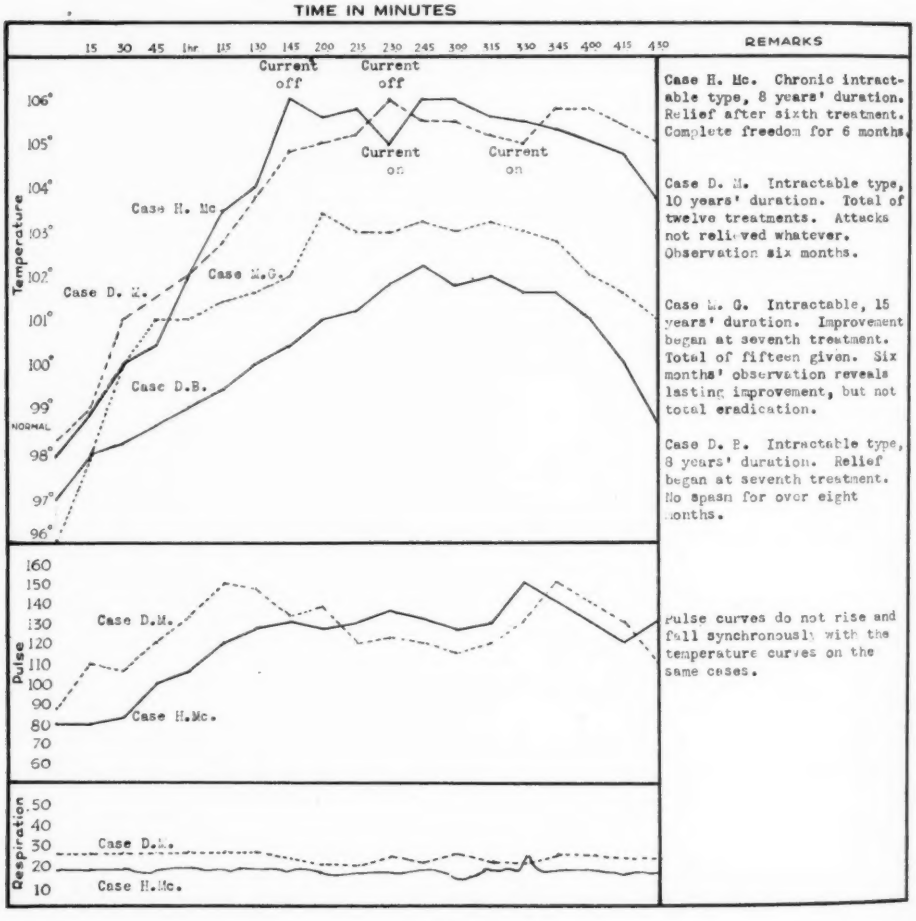
Method of Study.—We, therefore, did not select our cases, but chose fifty patients suffering from bronchial asthma in chronological order regardless of sex, age, type of asthma (acute,


TIME IN MINUTES
15 30 45 1hr. 15 130 145 200 215 230 245 300 315 350 345 400 415 430
REMARKS
Both patients hospitalized but not bedridden.
Temperatures taken every four hours were subnormal. Only the morning and evening readings are recorded.
Case M. G. following course of Electropyrexia now runs normal daily temperatures. See Graph II.
Temperature
106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
NORMAL
Sept. 24 Sept. 25 Sept. 26 Sept. 27 Sept. 28 Sept. 29 Sept. 30 Oct. 1 Oct. 2
8 AM 8 PM 8 AM 8 PM 8 AM 8 PM 8 AM 8 PM 8 AM 8 PM 8 AM 8 PM 8 AM 8 PM 8 AM 8 PM 8 AM 8 PM
Case J. W.
Case M. G.
Pulse
160 150 140 130 120 110 100 90 80 70 60
Case J. W.
Case M. G.
Bradycardia in Case M. G.
Respiration
50 40 30 20 10

Graph 1

Illustrating the subnormal daily oral temperatures of two cases of bronchial asthma observed over periods of nine days. Morning and evening temperatures only are recorded. Both cases following their course of ten hyperpyrexia treatments now maintain normal daily temperatures.

chronic, intractable), previous treatment or other associated factors. Every patient was treated with one course of hyperpyrexia which we arbitrarily fixed at ten treatments. Wishing to establish the proper time interval between each treatment, this group was divided into two parts, one sub-group receiving treatments once a week, the other twice a week. No attention whatever was paid to allergic manifestations, since any allergic therapy would veil the results obtained by the electropyrexia. Many of our patients, however, had been previously studied from the allergic standpoint and treated accordingly without benefit. A number of patients suffering from upper respiratory infections and mechanical obstructions such as enlarged tonsils and adenoids, as well as some patients with sinusitis, were treated for these conditions by the usual methods because the bronchospasm was so constant and severe that it was impossible to treat the patient with electropyrexia until some measure of relief had been attained. The greater number, however, were only treated with electropyrexia. In order to throw more light on the physiologic changes and to attempt to interpret conflicting reports in the literature, each patient was given a routine examination at the beginning, during and after the first treatment and



Graph 2

Illustrating the typical curves of two cases elevated and maintained at the higher temperature and two cases at the lower temperatures. Note the independence of the pulse curves to the high temperatures. The end clinical results are summarized.

again after completion of the course. We recorded and observed the following: weight, temperature, pulse, blood count, blood pressure, basal metabolic rate, alveolar carbon dioxide tension, vital capacity and McDonough colloidal bodies. A chemical analysis of the blood, urine and perspiration, including calcium, chlorides, serum bilirubin, nonprotein nitrogen, urea nitrogen, uric acid, blood, sugar and acid-base balance was also made. Following the complete course the entire group was observed for a period of from three to eight months.

In treating the bronchial asthmatic we believe we can obtain the same results with temperature below 105° F., maintained two to four hours, as with higher temperatures, providing we pay more attention to what appears to be an important factor, namely: a gradual rise of temperature instead of a rapid one. Therefore, we do not have to hospitalize a patient for the sole purpose of treatment since the treatment period is comparatively short. However, a department properly equipped with beds, technicians, nurses and attendants is a basic requirement, since electropyrexia cannot be said to be a simple office procedure, although the patients need not necessarily be kept overnight. *Graph 2* represents typical temperature curves for two cases of each group. *Tables 2* and *3* give a survey of the biochemical changes and clinical results of part of the group of fifty.

The actual technic and the types of apparatus most suitable for the production of electropyrexia have been thoroughly described in the current literature by Neymann,[10] Neymann, Feinberg, Markson and Osborne,[15] Osborne and Markson[16] and others.

Management of the Patient.—The only special preparation necessary consists of paying attention to intestinal elimination and having patients

Table 2

SHOWING BIOCHEMICAL AND LABORATORY FINDINGS IN NINE CASES BEFORE, DURING AND FOLLOWING ONE COURSE OF TREATMENTS

		Blood Counts			Blood Chemistry					Urine	
Patient		R. B. C.	W. B. C.	Eos.	Sugar—Mg.	Urea—Mg.	Alv. CO_2	Alk. Res.	S. G.	Urea—Mg.	Acidity—Deg.
H.M.—Male	Before	4,200,000	4,000	6	57	16.5	30	8.4	1017	12	27
	Height	4,800,000	9,400	5	87	12	20	8.2	1012		8
	After	4,800,000	9,500	4	95	12	25	8.2	1016	20	26
B.L.—Female	Before	4,800,000	7,600	10	80	20	45	8.6	1018	14	50
	Height	5,200,000	20,100	6	98	16.5	45	8.2	1003		10
	After	4,700,000	11,500	4	104	12	25	8.4	1018	18	18
E.M.—Female	Before	3,530,000	8,800	9	78	22	45	8.3	1013	10	18
	Height	4,500,000	11,400	5	96	16	45	8.3	1008		4
	After	4,800,000	9,000	7	102	12	45	8.2	1015	18	16
J.B.—Male	Before	3,960,000	9,400	14	90	23	25	7.6	1018	12	52
	Height	4,500,000	12,600	5	97	18	25	7.8	1003		5
	After	5,050,000	10,200	6	102	16	30	8	1025	20	18
R.J.—Female	Before	4,270,000	10,600	7	98	18	45	8	1019	10	34
	Height	5,130,000	14,100	5	107	14	45	8.2	1023		23
	After	6,120,000	11,200	6	106	10	45	8.4	1016	16.5	22
R.W.—Male	Before	2,800,000	8,600	14	72	21	35	7.8	1021	8	72
	Height	3,130,000	11,500	6	87	16	30	7.6	1017		20
	After	3,500,000	9,200	7	92	14	30	7.6	1017	16.5	11
B.L.—Male	Before	3,960,000	5,050	10	87	16.5	30	7.6	1021	14	33
	Height	4,000,000	11,400	7	96	13	30	7.8	1009		5
	After	3,900,000	9,300	7	97	11	20	8.2	1016	18	18
B.O.—Female	Before	4,270,000	4,500	20	68	18	30	7.8	1018	12	22
	Height	5,120,000	9,500	13	82	14.5	30	8	1013		17
	After	4,260,000	6,900	16	102	12	30	8.2	1016	18	15
R.M.—Male	Before	4,440,000	10,600	16	82	20	45	8.4	1019	10	
	Height	4,800,000	14,100	13	98	15	45	8.2	1022		
	After	4,300,000	8,800	16	106	15	45	8.2	1016	16.5	

void before beginning treatment. The patient is instructed not to eat any solid food for four to eight hours previous to the treatment. Temperature elevation is guided solely by rectal temperature, as mouth temperatures are unreliable, sometimes lagging as much as four degrees during treatment. Water is given copiously during the treatment and as the higher temperatures are reached an ice cap is applied to the head. While there is considerable discomfort during the period of high temperature, we, unlike many other workers, have not found it necessary to give sedatives. Neymann and Osborne,[17] in a recent publication, have shown some physiologic reactions to morphine, hyoscine and pilocorpine during electropyrexia treatments and discourage their use. In some of our cases the treatment actually aggravated or produced bronchospasm. In these instances we give epinephrine during the treatment. When the desired temperature is attained, the diathermic current is turned off, but the machine is left connected in case a further application of current is needed. When it is time to terminate the treatment the blankets are removed gradually and when the temperature has fallen to 99° F. the patient is taken out, given an alcohol rub-down, placed in a clean dry bed and allowed to rest a while previous to his departure.

Physiological and Biochemical Studies.—In reviewing the literature of other workers in this field it is apparent that there is need for more biochemical and laboratory study to aid us in determining how fevers obtained in this way produce their therapeutic effect. Most authors seem to agree that infection with malaria or the injection of foreign proteins for producing fever in the asthmatic would be uncertain and dangerous.

The reasons for clinical recovery or benefit after electropyrexia are not known. Theories have been advanced attributing this on the one

Table 3

ILLUSTRATING THE VALUE OF HYPERPYREXIA, REGARDLESS OF TYPE, AGE OR SEX, IN TEN CASES. THIS TABLE ALSO INDICATES HEIGHT, DURATION OF THE TEMPERATURE AND THE TOTAL NUMBER OF TREATMENTS TO A COURSE

Patient	Type and Duration	Temp. Peak Deg.	Duration in Hours	No. of Treatments	Results and Remarks
D.M.—35 Female	Intractable, constant with severe exacerbations; 10 years; adrenalin fast	105	5	10	Treatments aggravated attacks; no relief during following 4 mos. Suggestion of improvement at end of 6 mos.
B.L.—16 Male	Intractable, constant exacerbations; 8 years	105	4	10	No improvement for 2 mos., then marked gradual improvement. No spasms for past 90 days
H.M.—46 Female	Chronic, intractable, constant; 20 years	106	5	3	Stopped treatment. No improvement.
B.M.—18 Male	Chronic, periodical every three weeks; 2 years	104	3	10	Complete relief at eighth treatment. No return in 8 mos.
M.G.—53 Female	Constant, intractable, severe; 30 years	104	3	15	Relief almost complete during day. Some spasm at night, requiring 3 min. adrenalin for comfort. Four months' observation
L.T.—10 Male	Chronic; periodical attacks every 10 to 14 days	104	4	10	Complete relief for 4 mos. Eight months' observation
D.B.—7 Male	Intractable; constant since 2 years of age	104	2	15	Complete relief for 4 months. Recurrence of attack less severe than old form
E.W.—36 Female	Acute, periodical; duration 6 mos.	103	4	10	No bronchial spasm in 8 months
M.N.—10 Male	Chronic, periodical; 4 years	106	4	15	Complete relief for 3 months, followed by light attack. No spasm for past 3 months
G.S.—8½ Male	Chronic, periodical; 4 years	106	4	15	Partial relief only at end of 4 mos. Hypopituitary type. Tonsils infected. Father and mother are first cousins

hand to the action of heat alone, and on the other to changes in the autonomic nervous system and the endocrine glands. Fever is considered at length in books on metabolism,[18] but they offer very little help in solving the problem. We have, therefore, made biochemical studies with the hope of throwing more light on this subject. Most of this work is sufficiently extensive to be complete; some, to be sure, must still be considered as preliminary and we are at present making further studies.

Blood Counts.—Feinberg, Osborne and Afremow[5] found that the red blood count increased about 10 per cent and the white count as much as 100 per cent with some reduction in the eosinophil during fever. Leopold and Stewart[7] agreed that the white blood count was increased. Schamberg and Tseng,[19] using baths, came to the same conclusions and also noted an increase of the polymorphonuclears. Mehrtens and Pouppart[20] and Neymann and Osborne[3] have made similar observations, while Perkins[21] found very little variation in the red count, but the white count increased after the first treatment.

Complete blood counts were taken before the course of ten treatments was started in sixty patients between the ages of six and fifty-five years. The sexes were about equally represented. These original counts were made in the morning on a fasting stomach in order to minimize the factors of exercise and digestion. A second blood count was taken at the height of the fever and a third following the return of the temperature to normal. After a course of ten treatments and within one week following the last treatment the complete count was repeated. We can confirm the results of Feinberg, Osborne and Afremow.[5] The red count increases about 10 per cent during hyperpyrexia and the counts following the entire course show no notable change. The change in the red blood count is therefore only temporary. This is not true of the white count. Here there is an increase in the total number of cells during the individual treatment varying from 4,000 to 20,000. The polymorphonuclears increase 10 to 15 per cent, the eosinophils decrease 3 to 5 per cent approximately, though sometimes no change is found, while in five cases there was an actual increase of from 2 to 4 per cent. No change in the monocytes was observed. Let us analyze the nine typical cases of *Table 2*. In explaining these changes in the blood count the first reasonable conclusion might be that it was of a temporary nature and due to dehydration during the treatment. However, this is not a complete answer, since there is such a marked difference between the increase in reds and total whites. Secondly, based solely on dehydration, we should get a decrease in neither eosinophils nor monocytes, as is reported by Neymann and Osborne.[3] Following the entire course of ten treatments we still find an actual increase in the total white count. This is especially true of the group showing leukopenia from the beginning. Three such cases are included in *Table 2*. Here the white count increased as much as four thousand cells. In this connection we must not overlook the possibility of a close association between immunity and this sustained increase of white blood cells. This may explain the clinical results in the asthmatic.

Blood Sugar.—In a study of over 400 cases of bronchial asthma[11] we found a constant tendency toward a low fasting blood sugar in over 80 per cent of the patients. This varied between 57 and 95 mg. per 100 c. c. Knudson and Schaible,[22] Neymann and Osborne[3] and Perkins[21] reported an increase in blood sugar during the febrile period. Perkins states that this disappeared at the end of each treatment. Asthmatics were not included in these reports. Thirty of our patients varying in age from twelve to forty-five years showed fasting blood sugars below 90 mg. During and after a single treatment the blood sugar increased from 14 to 20 mg., but following the course of ten treatments, only three cases remained below 90 mg., while the others varied within normal limits. This might indicate a stimulation of the endocrine glands and of the sympathetic nervous system. In this connection it is interesting to note that the analysis of the perspiration following a treatment shows 19 to 27 mg. of sugar per 100 c. c.

Nonprotein Nitrogen, Urea and Uric Acid.—In a recent study of asthmatics we[11] found no changes in the nonprotein nitrogen and uric acid beyond normal limits, but an increase in the blood urea was often observed. In more than 90 per cent of the patients the urea content of the twenty-four-hour urine specimen was decreased. As a matter of fact, it is rare to find a patient whose twenty-four-hour urea output is above 15 grams according to the hypobromite method. During single electropyrexia treatments, Neymann and Osborne,[3] Knudson and Schaible,[22] Walinski[23] and Perkins[21] all report increases in the blood nonprotein nitrogen. Schamberg and Tseng[19] report no change. Again, these reports did not deal with asthmatics. We studied the nonprotein nitrogen and blood urea of eighteen adults before, during and after a single treat-

ment. There is a slight increase in the nonprotein nitrogen, but a decrease in the blood urea. This decrease averages about 4 mg. per 100 c. c. Following a course of ten treatments, the urea and nonprotein nitrogen returned to normal levels even in the case of patients showing an increase of the blood urea in the beginning. The decreased urea of the urine before treatment increased to a normal level following the entire course. The perspiration yielded 24 mg. of urea per 100 c. c.

Acid-base Balance.—This phase of the asthmatic syndrome is at present receiving much thought and discussion. Acidosis was considered a part of this syndrome by the majority of workers until Beckman[24] suggested that asthma was a state of potential alkalosis. This is still an open discussion. We selected one hundred and thirty-five cases and made carbon dioxide determinations, using both the van Slyke open and oil methods. All readings were within normal limits. The large majority showed low normals between fifty-two and fifty-six. Because these issues are still unsettled we felt that it would be desirable to use methods heretofore not utilized in an attempt to arrive at the truth. Two methods described by Marriott[25 26] were utilized. One consists of determining the blood alkali reserve; the second consists of measuring the alveolar carbon dioxide tension. These tests were performed on fifty asthmatics. Four revealed a slight degree of alkalosis. *Table 2* gives a survey of nine of them. The remaining forty-six tended toward acidosis. However, only ten were below a low normal acid range. These results combined with those obtained by testing one hundred and thirty-five cases with the van Slyke method lead us to believe that individual variations in either direction can exist in asthmatics but that the group as a whole tends toward at least a low-grade acid intoxication. The same tests applied before, during and after hyperpyrexia reveal individual fluctuations in both directions. With the exception of four or five, the readings stayed within normal limits and we are inclined to doubt that there is much change.

Blood Calcium.—In sixty-five cases, using Collip's modification of the Tysdale method, we[27] found that sixty showed a total calcium within normal limits. The diffusible calcium and inorganic phosphorus are often slightly altered. Hyperpyrexia does not produce any significant change.

McDonagh Colloidal Bodies.—Cameron[9] has reported, and our work confirms the fact, that in the asthmatic there is a decreased motility with clumping and precipitation of giant particles. Following hyperpyrexia there is a dispersion of both. Certainly we cannot overlook the fact that in artificial fever we are dealing with basic colloidal changes. *Fig. 1* illustrates these facts.

Urine.—One of the constant findings in the asthmatic is the hyperacidity of the urine, when collected and studied by the method of Mitchell.[29] This has been previously reported by us. During hyperpyrexia this acidity drops in most cases. We have observed a reduction of titre from 52° before to 4° after fever, using tenth-normal sodium hydroxide. Neymann and Osborne[3] report an increased concentration of the

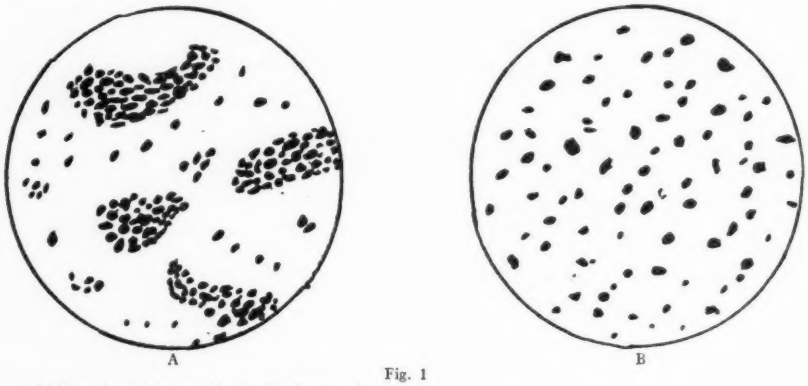


Fig. 1

McDonagh protein particles in blood serum in asthma.

(A) Before treatment, showing lack of motility, with precipitation of protein particles and giant cells.

(B) After treatment, showing dispension of both protein particles and giant cells.

urine with an increase of the specific gravity. This is exactly what we would expect. In thirty cases our results are confirmatory.

Perspiration.—The amount of perspiration collected during fever averages 24 mg. of urea per 100 c. c.; 23 mg. of sugar; 0.075 per cent of lactic acid according to the method of Strauss; and 33 to 97 mg. of chlorides.

General Metabolic Studies.—Attention has already been called to those clinical and laboratory findings, which lead us to believe that the bronchial asthmatic has a low metabolism and a sympathetic nervous system functioning at a low ebb. If these abnormal findings are corrected at the completion of a course of electropyrexia treatments, then we may assume that we have rendered some lasting relief. Such apparent changes are illustrated in *Table 2.*

Basal Metabolic Rate.—Our results confirm those of Neymann and Osborne[17] and others. The basal metabolism increases 7 per cent for each degree Fahrenheit. In those showing a low basal rate at the beginning the rate was definitely increased following a course of treatments, in some from —15 per cent to + 3 per cent.

Temperature and Pulse Rates.—Attention has already been called to the tendency of asthmatics to run low mouth temperatures. In many, a daily temperature of 97° is found; in some we observed daily temperatures as low as 96° F. Fifty cases were closely observed during the course of treatment in order to ascertain whether sufficient improvement would ensue to raise the metabolism and consequently the daily temperatures to a more normal level. Following the fourth or fifth treatment and at the completion of the course, all patients previously manifesting low temperature had reestablished normal mouth temperatures. It is interesting to note that some who had normal temperatures in the beginning and who later had a mouth temperature of from 99 to 99.2° F., often continuing eight to ten days after the course of treatment, finally dropped to a normal level. It has been reported that the heart rate fluctuates in direct proportion to the rise in body temperature. We find that in asthmatics there is no definite relationship between heart rate and the body temperature. The temperature may be raised several degrees without a corresponding increase in heart rate and the rate may return to its initial level while the body temperature still remains high. Further work in an attempt to explain this led us to manipulate our insulation in order to cause different temperatures of the skin. We find that the increase and decrease in heart rate is dependent upon the heating and cooling of the skin and is independent of the deep body temperature. *Graph 3* gives the curves of such a case, when the skin temperature is altered artificially. The pulse rate curves do not follow the curves of the rectal temperature. These skin temperature readings were made possible by the aid of Dr. Gasten LaBat, of New York City, who kindly placed his thermocouples at our disposal. Our explanation is that there must be thermal terminals in the skin which connect with reflex arcs leading to the accelerator and depressor nerves of the heart.

Blood Pressure.—The majority of our cases showed a lowered blood pressure in the beginning. A systolic pressure of 86 to 90 mm. was quite usual. Most workers have reported a rise of the systolic blood pressure with a fall of the diastolic as the rule during electropyrexia. In our fifty cases we did not find any regular or significant fluctuations. At the height of the temperature most of the cases show a slight rise averaging 10 mm. of mercury in the systolic and a fall averaging 10 mm. of mercury in the diastolic pressure. Some do not show more than 3 mm. fluctuation. It is possible that our tropical climate may have some bearing on these findings.

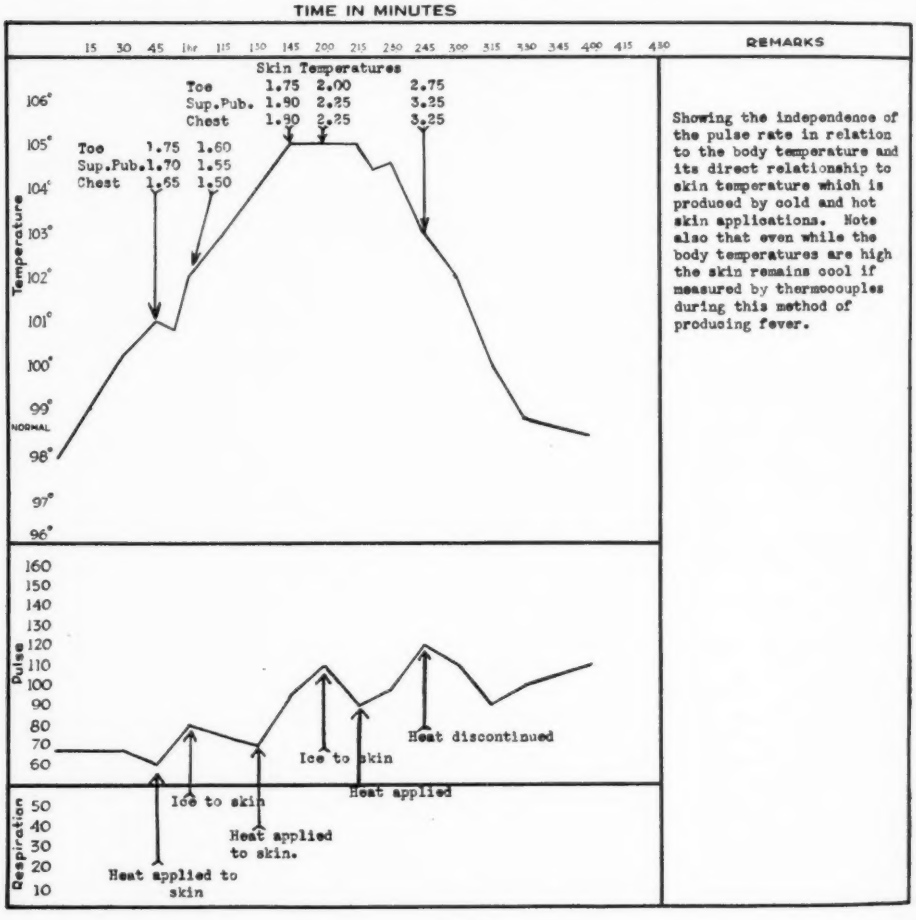
Weight.—In the beginning we hesitated to treat many asthmatics since they were poorly nourished, emaciated and half starved due to dietary factors which aggravated their attacks. We assumed that there would be a further loss of weight inasmuch as Neymann and Osborne[17] reported a temporary loss of as much as 8 kilograms during a single treatment. Of the entire series of fifty cases only two showed a loss of weight at the end of the course. All the others either held their original weight or gained, one case as much as eight pounds.

Vital Capacity.—The vital capacity is decreased in practically all chronic and intractable asthmatics. It increases as treatment progresses, providing the patient is improving clinically. Undoubtedly this increase is not directly related to hyperpyrexia.

Clinical Results.—We believed it might be logical to assume that if fever increases and accelerates the metabolism, it might prove a valuable clinical aid. This would become especially apparent if a larger series were treated more intensely. We selected our cases to include various ages, both sexes and all types of asthma. We

have attempted to eliminate as much as possible all influencing factors which might be responsible for clinical benefit and thus confuse proper clinical interpretation. Three cases were allergic to certain foods and before a reasonable degree of comfort could be secured for the patient it was necessary to eliminate the allergic foods from the diet. In two these allergic manifestations disappeared after the sixth and eighth hyperpyrexia treatment, respectively. In previous reports we[28] have called attention to the importance of pathology of the upper respiratory tract and its close relationship to bronchospasm. Infection in the upper respiratory tract may sometimes act as a trigger mechanism and in other instances as a factor in the general toxicosis. In about half of the group it was necessary for us to treat such conditions in association with hyperpyrexia. This may be used argumentatively by others to account for the results achieved. This is not our belief. Besides such an interpretation would not account for the favorable changes in the blood chemistry and body metabolism. These have not been observed by us heretofore when hyperpyrexia was not used.

We wish to emphasize that we do not consider it possible to evaluate any therapeutic procedure applied to bronchial asthma for permanent relief


TIME IN MINUTES
15 30 45 1hr 115 130 145 200 215 230 245 300 315 330 345 400 415 430
REMARKS
Skin Temperatures
Toe 1.75 2.00 2.75
Sup.Pub. 1.90 2.25 3.25
Chest 1.90 2.25 3.25
Toe 1.75 1.60
Sup.Pub. 1.70 1.55
Chest 1.55 1.50
Temperature
106° 105° 104° 103° 102° 101° 100° 99° NORMAL 98° 97° 96°
Showing the independence of the pulse rate in relation to the body temperature and its direct relationship to skin temperature which is produced by cold and hot skin applications. Note also that even while the body temperatures are high the skin remains cool if measured by thermocouples during this method of producing fever.
Pulse
160 150 140 130 120 110 100 90 80 70 60
Heat discontinued
Ice to skin
Heat applied
Respiration
50 40 30 20 10
Ice to skin
Heat applied to skin.
Heat applied to skin

Graph 3

Showing the independence of the pulse rate in relation to the body temperature and its direct relationship to skin temperature which is produced by cold and hot skin applications. Note also that even while the body temperatures are high the skin remains cool if measured by thermocouples during this method of producing fever.

until cases have been observed free from attacks over a period of at least one year. Our observations covering a period of eight months, therefore, must be considered as preliminary. We are still observing this group and we will report again after a further lapse of eighteen months. At the present time our results are as follows: out of the group of fifty patients, the asthma of three was undoubtedly aggravated by the treatment. In spite of this aggravation they remained with us for the entire course of ten treatments. Seven patients received no relief, but two of these stopped treatment at the end of the second and third treatment, respectively. The remaining forty all received clinical relief in varying degrees. Fifteen appear to have obtained lasting relief. Thirty-two have received clinical relief, but their biochemical and laboratory studies still show them to be potentially in danger and further observation will be necessary to determine whether their improvement is temporary or permanent.

GENERAL DISCUSSION

From this study one might tend to become over-enthusiastic. No doubt further carefully controlled work will give the information necessary for a true appraisal of the clinical value of hyperpyrexia in bronchial asthma. The important thing is not to be dogmatically prejudiced on the one hand and over-enthusiastic on the other. The entire field of electropyrexia is still in its infancy. In our opinion it undoubtedly will become one of our most valuable assets in the treatment of bronchial asthma, just as it has established itself in the treatment of central nervous system lues. We believe that it will be used as part of the therapeutic armamentaria and not as a treatment by itself. This line of reasoning is based upon the laboratory and biochemical studies which show that it has a tendency to increase general metabolism and reestablish normal physiological functions. As we have emphasized in former publications, underlying pathologic conditions must be present which produce these abnormal physiologic findings. Unless these are properly sought for and corrected it is unreasonable to expect permanent results from this type of therapy.

Especially interesting to us are the laboratory findings during the treatment, namely: the marked fall in the titratable acidity of the urine and the increase in its specific gravity. These occur constantly whether water is given copiously or withdrawn during treatment. Of equal interest is the decrease in blood urea, the increase in blood sugar and the quantitatively increased excretion of sugar, lactic acid and urea in the perspiration.

The other findings suggest a stimulation of the endocrine glands and of the sympathetic nervous system. Possibly new avenues of approach in these disorders may thus be opened.

We believe that ten treatments should be the minimum for a course. It would appear that twelve to fifteen are possibly still better, but apparently there is no improvement beyond this number. Two treatments a week are preferable to one a week. Some of our best results, however, have been obtained by weekly treatments. Temperatures maintained above 105° F. rectally have no advantage over those ranging between 102 and 105° F. Temperatures sustained for four hours produce results comparable to those extending over longer periods. In this connection we wish to emphasize the fact that we are reporting only upon bronchial asthma. With this technic the treatments can be carried on with ambulatory patients.

CONCLUSIONS

(1) The value of hyperpyrexia in fifty cases of bronchial asthma has been investigated. Practically all other therapeutic factors were eliminated.

(2) Clinical, biochemical and laboratory studies were carried out on all patients in order to determine the physiologic and biochemic changes associated with the treatment.

(3) Attention has been called to the clinical and laboratory data which indicate a lowered general metabolism in asthma. Improvement of these findings followed fever treatments.

(4) The clinical results indicate 82 per cent received definite benefit. Biochemical and laboratory studies, however, indicate that over 40 per cent of these were possibly only temporarily relieved. At present, after six months' observation, 32 per cent seem to have received a lasting relief.

(5) This report is limited to the actual time that our patients have been observed, a period of from six to eight months.

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DISCUSSION (*Abstract*)

Dr. Warren T. Vaughan, Richmond, Va.—Any attempts to improve the therapeutic armamentarium in a chronic malady such as bronchial asthma are worth while. I regret that the speaker in his series did not differentiate the intrinsic asthmatics from the extrinsic asthmatics. This has been the great source of trouble with the nose and throat men and their attempts to evaluate results of treatment. When one goes through the rhinologic literature on asthma one finds that the reported incidence of sinus disease in chronic asthma varies anywhere from zero to 100 per cent. The results of local treatment vary almost as much. This is because it is erroneously considered that all asthmatics are alike. Obviously the chronic asthmatic sensitive to house dust and relieved simply by the process of house dust avoidance or desensitization, is altogether different from the intrinsic asthmatic, not sensitive to any allergen with which he comes into contact from the outside. This is the so-called bacterial asthmatic. No statistics on the results of treatment of asthma can be of much use unless the cases are classified, at least to the extent of determining, by test, whether we are dealing with extrinsic causative factors or with intrinsic causes.

If I remember correctly, there have been three papers on diathermy treatment of asthma by Piness, by Leopold, and by Feinberg. The first two decided that the treatment was not successful, while the third report was favorable. Dr. Phillips' report would make the odds even on both sides.

I hope that as Dr. Phillips continues this work he will take advantage of the opportunity of classifying his asthmatics from the etiologic viewpoint.

Dr. Edward W. Griffey, Houston, Tex.—There is no more important field for clinical research that presents itself today than bronchial asthma. The essayist referred to the "trigger phenomena." When I was doing otolaryngology I observed many remissions and the absence of asthmatic symptoms over long periods following the resection of nasal polyps in asthmatic patients.

Dr. Charles T. Stone, Galveston, Tex.—As Dr. Phillips stated, it has been frequently observed that when an asthmatic develops an acute febrile disease the symptoms of his asthma may disappear, at least temporarily. According to the best information, this improvement appears to be a non-specific action and not an immunologic one. The specific skin reactions given by the patient may diminish or even disappear entirely at the height of the fever, but as a rule they reappear as strongly as before within a few days after its defervescence. This status, that is, sensitization but with freedom from symptoms of asthma, may persist for many weeks or even months. It may be stated, therefore, that the effects of fever in producing improvement in asthma result from causes other than desensitization.

Any therapeutic procedure which completely relieves a fair number of asthmatics over a period of months, and diminishes considerably the number and severity of attacks in another large percentage of cases as Dr. Phillips has reported, is a distinct addition to the existing methods of treatment. The fact that it is wholly nonspecific does not constitute a valid argument against it. Patients with neurosyphilis improve strikingly under malaria therapy, and some cases of infectious arthritis of obscure etiology are benefited to a marked degree by the fever that follows the administration of typhoid vaccine intravenously; yet in neither instance is the treatment in any sense specific. Even in infectious processes where chemotherapeutic measures are followed by a measure of success, one cannot invariably produce *in vitro* results that are comparable to those occurring *in vivo*. In all of these instances it has been assumed that a state of "altered tissue reactivity" has been produced and that this accounts for the improvement. The fact that so large a number of invasions of the body by toxic and bacterial substances results in fever led

the late Dr. William H. Welch many years ago to say that such a rise in body temperature "has not been developed to so wide an extent and is not retained with such pertinacity without subserving some useful purpose," and only recently the Council on Physical Therapy of the American Medical Association emphasized that "the treatment of disease by means of hyperpyrexia is now established."

Extensive biochemical and physiologic studies of changes resulting from hyperpyrexia induced by physical means have been and are being undertaken by different workers. These deal with a large variety of observations, for example, variations in the temperature in different parts of the body, velocity of the blood flow, leukocyte counts, red cell sedimentation rates, basal metabolic rates, blood serologic studies, and the chemical changes in sweat, blood, urine and gastric contents. Out of this mass of investigation have come fairly uniform findings of alterations in many of the body functions during fever. Usually they are of but temporary duration and return to normal as the fever declines. It is, therefore, difficult, if not at present impossible, to say with certainty what it is that artificially induced fever does which causes the improvement not only in asthma, but in general. Despite the fact that the existing knowledge of the whole subject is largely empiric, it in no wise detracts from the usefulness of the method, but on the other hand should stimulate additional clinical investigation of the problems involved, along the thorough and painstaking lines followed by Dr. Phillips and his associates.

Dr. D. H. Hotchkiss, Jr., Houston, Tex.—Unquestionably artificial fevers will become a most useful adjunct in the treatment of selected cases of asthma, but in just which cases remains to be discovered.

I regret that in this series of patients no record was made of intradermal skin tests and that no division was made into certain clinical groups such as those having acute paroxysmal asthma and those suffering with the chronic asthmatic state. Observations made in the handling of several hundred asthmatics in a practice limited to allergy force me to believe that paroxysmal asthma is an uncomplicated protein hypersenstiveness, while the chronic asthmatic state has a more complex nature. Comparative statistics of the clinical results in these two groups will be most interesting.

Asthma is a syndrome caused by a specific antibody-excitant reaction. These antibodies have been proven to exist by their passive transfer to the skins of non-sensitive recipients. The ability to produce them is a dominant hereditary characteristic. Positive local reactions can be obtained in the large majority of asthmatics by activating these antibodies by the injection of an extract of the exciting agent into the skin. Such tests done before and after the course of treatments of hyperpyrexia would have given valuable evidence concerning the effect on the specific antibodies. It is difficult for me to see how this heat could have destroyed or rendered inactive either these antibodies or the cells to which they were attached. Unless at least one of these ends is obtained the patient will remain a potential asthmatic. Skin tests also would have pointed out the specific etiological agent and this knowledge would have helped in allowing for variations in exposure to it when considering the final results. For example, an uncomplicated pollen-sensitive case may not have been exposed to a sufficient quantity of the pollen to produce an attack during the period of the observation. The three food cases mentioned comprise a series that is too small from which to draw any conclusions. Rowe and others have shown that complete withdrawal of an offending food from the diet for a period of several months results frequently in a loss of the clinical hypersensitiveness.

While all bronchial asthma is caused by hypersensitiveness to some specific protein, many outside factors can influence the symptomatology and treatment. Of major importance among these factors is infection in the respiratory tract. In Houston the climate is so temperate that some pollens, notably the grasses, remain in the air throughout the year and it is so damp and changeable that respiratory infections are extremely prevalent. Experiences under these conditions have given me the opinion that the proteins of these infections alone never cause asthma. When such infection is added to an underlying hypersensitivity, however, it changes the picture from the paroxysmal type to the chronic asthmatic state where specific treatment is of no avail. This infection may be very insignificant in itself and may require demonstration by methods that will visualize an hypertrophic mucous membrane. Control of this pathology (in the sinuses by local treatment or by conservative or radical surgery and in the bronchi by x-ray therapy to enlarged hilus lymph nodes) removes the factor that lowers the threshold of the patients' tolerance and permits specific treatment to become effective. Since fever, a defense reaction of the body, is rarely seen in pure asthma and almost invariably accompanies infections, I am inclined to believe that artificial hyperpyrexia will produce valuable lasting effects only in those asthmatics who have a concurrent infection complicating their condition. For this reason I shall be most interested in seeing the comparative results on these cases grouped as suggested.

This report is most excellent and the essayists should be given our sincere thanks for the scope they have covered in their investigation. All of us who are interested to any degree in asthma will be waiting anxiously for the final report of this work.

Dr. Phillips (closing).—The set-up for proper work in hyperpyrexia, if it is to be given safely, involves much time and expense in equipment and trained personnel. For this reason the primary object of this entire work was to expose the method to a thorough test and if the results indicated merit, to encourage other workers to invest their time and money; if the method manifests little or no value, to sound the warning and perhaps prevent others from further loss of time and expense. We, therefore, applied the method to a large group of asthmatics, including both sexes, acute, chronic and intractable types, varying in age from six to seventy-two years.

Dr. Vaughn expressed the view that this paper, together with the three papers published by other workers, might leave one still in a quandary, since two report favorably, one unfavorably and one indifferently as to the value of the method. We do not accept this, for the simple reason that in the light of what our work has revealed none of the papers previously published on applying hyperpyrexia to bronchial asthma is justified

in drawing any definite conclusions. In the first place, with the exception of Feinberg and his group, who treated seventeen patients, none of the other workers treated over two or three cases. Secondly, they all applied the method to old chronic intractable cases which, in itself, is also not a true test as to the value in general. Furthermore, with the exception of one case, none of the cases in the entire three groups received more than two or three treatments, a very significant factor, since our work has constantly shown that no results can be expected from less than six to eight treatments and we have established a minimum of ten as a course.

Reference to the cases of Miller and Piness will reveal that they utilized oral temperatures. Please refer to our graphs, which show a difference of as much as 4° F. between oral and rectal temperatures during hyperpyrexia. Neymann properly decries the use of oral temperature as a guide during hyperpyrexia and we believe that he is also correct in his view that many workers have not yet properly mastered the technic of inducing and sustaining the temperature.

These cases have been placed on the inactive observation list for six months. At this time there appears to be a very definite favorable result in about 33 per cent. We do not believe that the permanency of any therapeutic measure in bronchial asthma is thoroughly tested until cases have passed at least one year with complete freedom from bronchospasm. This demand alone might materially alter our present conclusions when that time has passed.

THE USE OF THE ROENTGEN RAY IN INFECTIONS*

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Occasionally papers have appeared in various medical journals on the subject of radiation therapy in inflammations. But for the most part radiologists have so emphasized the treatment of malignancy and skin lesions that the medical profession generally has little knowledge of what great assistance can often be furnished by roentgen ray therapy, and occasionally in special conditions by radium therapy, in the treatment of infections.

Our own, may I say, deficient enthusiasm in pointing out the advantages of this type of treatment can certainly not be explained on the basis of poor results. On the contrary, these results are usually good, frequently little short of magical, and never harmful, when radiation is properly applied. Our failure to emphasize the value of such therapy is, I believe, in part due to the difficulty in this age of scientific medicine, of placing it on a thoroughly rational basis. Another and more important reason is the rather obvious futility of trying to persuade the medical profession to turn to radiation when it is able to handle the large majority of inflammatory processes fairly satisfactorily without it. In other words, our natural tendency has been and is to wait for those cases in which the physician and surgeon already know that they need our assistance. This, of course, limits our field in large part to the more chronic and often less satisfactory cases.

While radiation therapy can give a very good account of itself in these chronic and subacute cases, it is in the acute lesions that the most spectacular results are obtained. Often a single treatment is all that is required in acute inflammation.

In the pyogenic types of infection as exemplified by furuncles, otitis media, mastoiditis, frontal and maxillary sinusitis, cervical lymphadenitis, acute parotitis, and so on, many authors[1 2 3] have reported most excellent results. When the treatment is applied early, the course of the inflammatory process is usually greatly changed. Very often the lesion does not go on to the suppurative stage, but instead the inflammation rapidly subsides. Sometimes there may be an initial increase in pain and discomfort. A recent case of acute cervical lymphadenitis displayed this phenomenon during the first 24 hours, although by the end of 48 hours the lesion had entirely disappeared. Usually, however, pain and soreness gradually begin to leave in the first few hours after treatment. In cases which go on to suppuration due to late treatment or other causes, the area of inflammation commonly becomes much more localized, the suppurative process is usually hastened, the symptoms are ameliorated, and the course of the disease is distinctly shortened. Acute parotitis, an occasional surgical complication particularly in colon operations, deserves especial emphasis on account of the high mortality which it brings about in association with the operation. Here radium is used in preference to the x-ray because it can be applied without moving the patient. Rankin and Palmer[4] report that when the treatment is applied early, there is usually a rapid subsidence of the inflammatory process with suppuration occurring only occasionally. Needless to say, there is also a marked reduction in mortality.

When we deal with acute infections of the non-pyogenic type, radiation therapy does not

*Read in Section on Radiology, Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

give as good an account of itself as in the conditions just mentioned. The beneficial effects are, however, sufficient in amount and frequency of occurrence to warrant a trial. This is especially true, since in such conditions as erysipelas and whooping cough other types of therapy offer very little. Occasionally in both conditions spectacular improvement may follow one treatment. At other times the improvement may be slight. If so, a repetition of radiation after four or five days will not be likely to contribute much more. Even though the improvement in the paroxysms in whooping cough does seem slight, it may spell the difference between life and death in very young children and babies who have a very severe type of the disease. Under this heading the treatment of unresolved pneumonia might also be mentioned. Several authors have reported a promptly initiated resolution following roentgen therapy. A few favorable results have also been reported[5] in earlier phases of this disease. Kelly,[6] in a small series of cases, reports some remarkable results in the treatment of gas gangrene when the x-ray was used in conjunction with other forms of treatment.

As stated previously, the subacute and chronic cases, in which other types of therapy have failed, furnish a large percentage of the infectious conditions submitted to us for treatment. Naturally the results are attained much more slowly than in the acute cases and only after the application of a series of treatments. One of the best examples of this type of case is the cervical adenitis of tuberculous etiology. Here roentgen therapy began to pave a way for itself even in its infancy. As technical improvements made more accurate therapy possible, it has gradually displaced surgery. For many years it has been the treatment of choice in this condition. In a recent article on roentgen treatment of cervical adenitis in which the tuberculous form is especially emphasized, Pfahler and Kapo[7] give an extensive review of the literature and report a large series of cases with excellent results.

Numerous other chronic processes have been treated with roentgen therapy with many favorable results reported. They include anthrax, actinomycosis, nephritis, trachoma, gastric and duodenal ulcer, pelvic inflammations, chronic sinusitis and bronchitis, chronic osteomyelitis and rheumatic heart disease.[8] Recently Heinz Langer[9] reported a large series of cases of arthritis treated by roentgen therapy with very good palliative results. My quite limited experience with his method would lead me to commend it heartily.

Judging from the reports of beneficial and ofttimes curative results in a wide range of acute and chronic inflammatory conditions, one must conclude that radiation therapy is worthy of honest consideration as an aid in the treatment of the majority of infectious processes. I am forced to this conclusion in spite of the hesitancy which we should all exercise in promulgating anything like a panacea, and with due regard for the prejudices naturally aroused among physicians when a wide application is claimed for any therapeutic agent. Unfortunately, as is the case with many therapeutic successes, it is impossible to explain adequately the modus operandi which brings about these favorable results.

Occasionally, radiation therapy seems to lower the resistance of the tissues to infection. This usually occurs in the case of the heavy radiation incident to the treatment of malignancy. A good example of this is the occasional lighting up of a latent pelvic infection while a cancer of the cervix is being treated. While such cases are rare, one experience cannot fail to make an indelible impression on the mind of the radiologist.

In many cases, however, fairly heavy radiation seems to exert a decidedly beneficial effect on infection. In rather late carcinoma of the cervix with accompanying infection, it has been the policy of radiologists for several years past to apply external roentgen therapy first in the usual heavy dosage, partly for the purpose of clearing up the infection prior to the administration of radium. The rather heavy general radiation reaction of the x-ray seems highly desirable in the presence of the infection, but not the heavier local devitalizing action of the radium. Several years ago I treated a chronic osteomyelitis of the femur with roentgen therapy under the mistaken diagnosis of osteogenic sarcoma. The results were decidedly beneficial even though I had naturally applied quite heavy dosage. A considerable period of time elapsed before I learned why my radiation therapy produced such unusual results in this supposedly malignant bone tumor. When one compares the effects of the heavier doses with those of mild dosage which we intentionally use in treating simple infection, he must conclude that they have much in common. In other words, it hardly seems proper to speak of stimulating

doses in an attempt to contrast the milder with the heavier dosage.

Some years ago we were very prone to explain the action of certain specific drugs as due to a special toxic effect directly on the organism in question. We have since learned that the action is much more complicated than that. Roentgen rays and the *gamma* rays of radium of themselves do not possess any bactericidal properties. It does seem evident, however, that they must enhance the power of the tissues to destroy micro-organisms. Webster[10] has recently cited some experiments which tend to prove this. Desjardins,[11] in an excellent article which carries an extensive bibliography, offers a very logical explanation of the effects of radiation in infection. He points out that there is a rapid breaking down of large numbers of the radiosensitive lymphocytes common to inflamed areas and that this is coincident in time with the rapid improvement which commonly takes place in early acute cases. He thinks the breaking down of these lymphocytes probably causes a more rapid liberation of antibodies. Important links in his argument are the facts that radiation is most beneficial during the infiltrative stage and that moderate doses are sufficient or even preferable to large doses.

As to the technic employed, there seems to be a rather common agreement among radiologists that only moderate dosage should be used. In different hands this may vary between 15 and 50 per cent of a skin erythema dose (300 r), rarely higher. My own tendency is to keep in the neighborhood of a 15 to 25 per cent skin erythema dose and to use a somewhat smaller dosage in children than in adults. For a long time I have labored under the impression that a larger total dosage could be applied without danger of skin changes if some copper filtration were used instead of only aluminum. Accordingly, this has always been my policy, although in the majority of cases the total treatment will naturally be well within the margin of safety. It would appear that recent trends in radiotherapy substantiate this idea. The filtration and voltage should, of course, be varied in accordance with the depth of the area to be treated. When dealing with tuberculous lymphadenitis it is always necessary to give a series of several treatments. In my opinion these should not be applied much oftener than 7 to 10 days apart. In other chronic or subacute conditions it may seem advisable to repeat the treatments every 4 or 5 days, but I believe it is questionable whether it is ever worth while to give more than 3 or 4 treatments in a single series in such cases.

The following cases are cited in an attempt to emphasize some of the points which I have endeavored to bring out:

Case 1.—Master J. McG., aged 3½ years, was referred for roentgen therapy because of an acute cervical lymphadenitis involving both sides of his neck. Thirteen days previously he had developed a sore throat accompanied by croup and fever. Two days later the cervical lymph nodes on the right side became sore and began to swell. Shortly after this the inflammation also involved the left cervical nodes, but the swelling was not marked and there was little discomfort on this side until a day or two before he appeared for treatment. From that time on the swelling rapidly increased until, if anything, this side was more prominent than the right.

Examination revealed tumors about the size of small eggs in the upper cervical region on both sides of the neck. They showed no evidence of beginning suppuration. The patient's temperature was 101°.

Roentgen therapy was applied to both sides of the neck and repeated five days later. On the left side where the inflammation was of rather short duration, the swelling rapidly disappeared after the first treatment. In the case of the right side, where the inflammation appeared first, the tumor disappeared much more slowly and it was nearly a month before it had entirely gone. No suppuration occurred on either side. This one case seems to demonstrate the advantages of early and the poorer results of late treatment.

Case 2.—Master A. O., aged 4 years, was referred for treatment in December, 1930, because of deafness, chronic cold and cough. His tonsils and adenoids had been removed in September, 1929, but by winter the pharyngeal tonsils had again hypertrophied so that once more he was compelled to breathe through his mouth. When summer arrived, mouth breathing was less noticeable, but after the cooler weather of the fall appeared it again became more evident. In November, 1930, a month before I saw him, he caught cold after which mouth breathing became very pronounced and marked deafness developed.

The child was very pale and acted as if he felt much below par. Examination of his chest revealed evidence of bronchitis. Unfortunately no x-ray pictures of his sinuses were taken, but it seemed quite evident that he presented a fairly typical picture of what Wasson[12] calls "bronchosinusitis."

A series of roentgen treatments were applied over the mediastinum and also directed toward the pharynx and sinuses from each side of his face for a period of about seven weeks at week and two-week intervals. At the end of this time his hearing was restored, his cough had disappeared and he showed no signs of cold. Six weeks later he looked the picture of health. Two years later the mother reported that there was still no evidence of the old trouble.

Case 3.—Mr. W. T. R., aged 28, in October, 1933, began to have pain near the angle of his jaw on the right side. A little later a tumor mass appeared a little in front of the right parotid gland. In November

the lesion was opened and some necrotic material removed. The incision was slow to heal, but eventually it closed, leaving a small tumor which remained stationary for a few weeks. In January, 1934, the tumor began to increase in size. It became somewhat painful and took on a definitely inflammatory appearance. The last of February, the patient was referred to me for my opinion and for treatment.

Examination revealed what was evidently an inflammatory tumor 1½ inches in diameter situated about 1½ inches posterior to the angle of the mouth on the right side. Near the middle of the tumor was a healed scar. The skin around the tumor was mildly hyperemic, but the mucosa adjacent to it on the inside of the mouth appeared normal.

Five x-ray treatments were applied over an area slightly larger than the lesion at intervals of seven to ten days. Three days after the first treatment the lesion broke open and discharged some pus, following which it gradually decreased to about one-half its original size. Shortly after the fourth treatment it again broke open and discharged. In about two weeks the tumor and inflammation had entirely disappeared.

Case 4.—Mr. B. M., aged 40, was referred to me for treatment in October, 1930, with a diagnosis of carcinoma originating in the upper right primary bronchus, a diagnosis in which I later concurred. His first symptoms had been ushered in three months before with a severe coughing spell, at which time he had pain in his upper right chest. Six weeks of antisyphilitic treatment had been given him with no apparent effect on the tumor. At the time I first saw him his chief symptoms were loss of strength, a dry rasping cough and an exceedingly foul breath, much like gangrene of the lung. X-ray picture revealed a large tumor shadow projecting from the upper mediastinum, right side. Since he was a charity patient, I did not ask for a bronchoscopic examination.

Three courses of moderately heavy roentgen therapy were applied at six to seven-month intervals in the hope of some palliation. The patient insisted that he was gradually improving, but little change was noted in the lung tumor on fluoroscopic examination until seven months after the last course of treatment. At this time the patient informed me that he was almost free of his cough. His breath no longer exhibited the previously foul odor and fluoroscopic examination showed the tumor had disappeared.

It seems very evident that we were dealing with some infectious process from the first, which fact should have been suggested to us by the extremely foul breath. The rather heavy roentgen therapy produced no discernible deleterious effects, but instead seemed beneficial.

Case 5.—Master J. S., aged 13, had a lymphangioma involving a large part of the calf of his right leg, which had been present since birth. Although various physicians had been consulted, no one had suggested radiation treatment, a treatment which he should have received for this condition in infancy. In early June, 1932, he was taken sick with scarlet fever and almost immediately infection, probably streptococcal in type, developed in this lymphangiomatous area and continued after the other evidences of scarlet fever had vanished. For a time this inflammation seemed to improve, but after a few weeks it reached a fairly stationary condition with mild recessions and exacerbations accompanied by some temperature.

In September, 1932, the patient was referred for radiation therapy. At this time almost the entire posterior part of the right leg from a little below the knee to a little above the ankle exhibited all of the cardinal signs of inflammation and, in addition, marked induration was present. The first treatment, which was a little less than a 25 per cent skin erythema dose, produced a marked febrile reaction. Accordingly, the next treatment was reduced to less than 15 per cent skin erythema dose and never thereafter was it increased much above that figure. The treatments were applied every five to seven days for a period of 10½ weeks alternately on two separate areas. At the end of that time the inflammation and fever had entirely disappeared, but the lymphangioma of course remained, with doubtless more fibrous tissue formation than was present before the scarfet fever. X-ray picture taken some months later shows a heavy fibrous tissue network in the subcutaneous region of this leg.

Case 6.—Master E. L., aged 3 years, had been sick for eight or nine weeks with what was thought to be a paratyphoid B infection. The week before he was brought to me for treatment he developed a pericardial effusion and exhibited evidence of an inflammatory process in the right hilus region. His condition was so desperate that radiation therapy was looked upon as a last resort.

Five roentgen treatments of moderate dosage (15 per cent to about 20 per cent skin erythema dose) were given over a period of a month with considerable benefit, resulting, as shown, by marked reduction in temperature and better appetite, and that, too, with an attack of measles intervening. Since then the child has been up and around part of the time, although he continues to run an intermittent temperature and obviously has a crippled heart. Occasionally some further x-ray treatment has been given. Always there is a definite subsequent evidence of benefit, but the disease still remains as a low-grade process. We shall evidently have to list this case as a failure as far as a cure is concerned, but it at least lends us hope that we may attain better results in the future.*

The few cases cited have covered a fairly wide range to illustrate the important place which radiation therapy should assume in the treatment of infections. Unfortunately, physicians and surgeons generally seem to look upon it at the present time as something to be tried after other remedies have failed instead of at the beginning of inflammation, when it offers most. It is very evidently our duty to put greater emphasis on this branch of radiation therapy. By so doing the practice of medicine will be advanced and the public better served.

*Six months after the above was written the child looks well and apparently feels well, as he exhibits the normal urge to play. He has had no temperature for several months. Skiagraphic examination, however, still reveals a very large heart shadow.

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DISCUSSION (*Abstract*)

Dr. C. P. Rutledge, Shreveport, La.—I should like to mention an infection in which I consider the x-ray of untold benefit. This is erysipelas, especially when it attacks the face and scalp. Not very long ago the members of the medical profession in my district sometimes as a last resort consulted the radiologist in handling these cases, but now my close associates ask for radiological consultation immediately. In erysipelas of the face and scalp our experience has been that x-ray therapy is the treatment of choice and saves many lives.

Dr. C. H. Heacock, Memphis, Tenn.—I believe that radiation therapy is now the method of choice in early furuncles, carbuncles and postoperative parotiditis. Complete subsidence of these infections without suppuration is the rule. Radiation should also be given in late cases of furuncles and carbuncles, because the course of the infection is shortened. Even if suppuration does occur, there is much less scarring. A furuncle, especially if it is situated on the back of the neck or the axilla, is usually only one of a series when treated surgically. One of the great values of radiation therapy in these situations is that it prevents another boil from developing in the neighborhood and breaks the chain of the infection.

When the infection is due to the streptococcus, radiation therapy may not be spectacular, but it is usually very effective. This was well illustrated in a case I saw recently in which all the subcutaneous tissue of the scalp was involved. Multiple incisions had been made and several blood transfusions were given without checking the course of the disease. The patient's condition was critical when I was asked to see him. Two x-ray treatments were given on successive days. The subsidence of the infection and the improvement of the patient was almost miraculous and he was discharged from the hospital on the third day after the last treatment. *Streptococcus hemolyticus* had been cultured from the wounds.

Recently I have seen two or three papers on treatment of gas bacillus infection with x-ray. I have had the opportunity to treat two patients. Both were treated after the infection was well advanced and all hope for recovery had been abandoned. In one patient the gas had infiltrated the tissues around and above the shoulder joint and no amputation had been done, although the tissues had been laid wide open by surgical incisions. The patient was given half a full dose of filtered radiation the first day and showed improvement. One-fourth of a full dose was given on the second and third days with full recovery. The second patient had had one amputation for gas gangrene when I was first called to see him. Gas had infiltrated the tissues above the point of amputation, which was in the middle of the arm. He was treated at once, using the same factors as were employed on the previous patient, but later on the same day a shoulder amputation was done. On the second day the gas infection had spread into the neck and a second treatment was given, but the patient expired a few hours later.

It is highly desirable that the value of x-ray in infection with the gas bacillus be more widely disseminated. The roentgen ray is not only very valuable in the early diagnosis of the condition, but it offers great value in its treatment. General and orthopedic surgeons have nothing to offer except mutilating operation and I believe, when they have become universally acquainted with the radiation method of therapy, they will be very willing to refer these patients.

Dr. Fred M. Hodges, Richmond, Va.—I have enjoyed Dr. Myers' paper, but feel that we should be extremely conservative in our claims.

There is no doubt that irradiation is the treatment of choice in furunculosis, erysipelas and parotitis. It is also of great value in the treatment of infection on the face, especially infections around the upper lip, nose and around the eyes. In the large majority of cases it is worth a great deal in the treatment of carbuncles. The doses should be small since as good results seem to be obtained with small doses and there is no danger whatsoever of injury to the skin.

END RESULTS OF LEG LENGTHENING*

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and
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Since the publication of Abbott's paper in 1927 on operative lengthening of the tibia and fibula, the procedure has been carried out in a number of orthopedic clinics. Many variations in technic have been tried and numerous types of lengthening apparatus devised. As might be expected from such a formidable procedure, the end results as a whole have not been entirely satisfying.

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The cases included in this report are taken from the services of Dr. H. A. Durham at the Shriners' Hospital for Crippled Children, Shreveport, Louisiana. They extend over a period of seven years, totaling 17 in number. Fifteen were lengthenings of the tibia and fibula and two were of the femur. The subjects for the procedure were carefully selected. All of the cases in this series had shortening resulting from infantile paralysis. No children under twelve years of age were selected. The minimum shortening of the affected leg, in all cases chosen, was one and one-half inches. None were selected who did not have at least fair muscle control of the thigh and leg. Two of the cases selected previously had had arthrodesis of the knee. These, while not considered ideal, were lengthened because of their extensive shortening, and we are convinced that the end results justified their selection. All cases are of sufficient duration to demonstrate conclusive end results.

The operative procedure used is similar to that described by Abbott. First an incision about four inches long is made directly over the outer border of the fibula in the lower third. The periosteum is stripped and the bone is then split over an area of about three inches with a bone saw. The anterior half is then divided at the lower end of the saw cut, and the posterior half at the upper end. The periosteum is divided through half of its circumference in two places on each side of the bone, the cuts not being opposite on the two sides. The wound is then closed with interrupted sutures. The lengthening pins are then drilled through the tibia, one about two inches above the internal malleolus and the other below the tibial tubercle. A slightly curved incision is made over the anterior aspect of the leg with the convexity outward. The periosteum is split longitudinally for about five or six inches and stripped from the entire circumference of the tibia. Two parallel saw cuts are then made, one on the medial and one on the outer sides of the bone respectively. These cuts are about four inches long and are so placed as to divide the bone into equal halves. The osteotomy is completed by dividing the posterior half of the tibia at the upper end of the saw cut and the anterior half at the lower end. The periosteum is then divided through half of its circumference in three places on each side of the bone, the cuts not being placed opposite each other. In addition to this the interosseus membrane is completely divided at the lower end of the wound. A ligature of No. 2 chromic catgut is then tied around the bone at the upper and lower ends of the cut to prevent displacement of the fragments.

Except in cases of calcaneus deformity the tendo achilles is usually lengthened by the Hoke method. Before the tibia and fibula are lengthened, the periosteum is closed with interrupted sutures of No. 0 chromic and the skin with interrupted sutures. A puncture wound is then made in the skin directly over the anterior border of the tibia at about the middle of shaft into which the stabilizer pin is inserted. Wounds are dressed, perforated squares being used over pin heads, held in place by strips of rubber tissues hooked over pin ends on front and back. The incisions are dressed separately without drainage and dressings are wrapped with sterile sheet cotton over which is applied a gauze bandage. The lengthening apparatus is then applied and the leg suspended. Lengthening is started on the fifth day. The leg is lengthened from one to three-sixteenths of an inch each twenty-four hours, depending upon the patient's tolerance. The incision over the tibia is dressed in three weeks. The dressings over the pin holes are not disturbed until the apparatus is removed, unless there is some very definite reason for changing it.

The lengthening apparatus used was designed by Dr. Durham. It consists of two extension devices made of tubing threaded on the outer side with a pin holder at each end. The upper pin holder is stationary, the lower one screws onto the tubing, which is swiveled at the upper end. This allows the lower segment to be rotated and thus produces extension by moving the lower pin downward. Two stainless steel pins five thirty-seconds of an inch (5/32) in diameter with drill points are used to attach the extension device to the tibia. The suspension portion of the apparatus consists of two three-eighths inch (3/8) steel rods which are passed through the hollow extension tubes. These bars fit into a half ring above and are locked in place with set screws. The lower ends of the bars are held with a slotted bar which is clamped tightly to each of the side bars. When all screws are tightened, the entire apparatus is rigid, preventing any twisting of the bone fragments. At the top of the upper pin holder is a cap screw, which carries a slotted arm. These arms are crossed over the upper fragment of the tibia, and are fastened together at the desired position with a hollow bolt, through which passes the stabilizer screw. This in turn fits into a socket in the upper end of the stabilizer pin. When screwed down to the desired tension this prevents any

anterior bowing of the tibia and keeps the fragments at all times in close apposition. On each extension tube is a scale graduated to sixteenths of an inch so that the amount of extension can be easily read each time the device is lengthened.

The time required for the lengthening varied from 15-25 days. After lengthening was completed the apparatus was left on from 4-5 weeks. Following removal of the apparatus a plaster cast was applied to the leg and the patient was then allowed up with crutches. A walking caliper was applied in all cases by the end of the month. The bones were well regenerated in from 6-8 months. In none of the cases was there any lateral displacement or inequality of length of tibia and fibula.

Complications.—There were no infections in any of the lengthening wounds.

Two cases had mild infection of the upper pin holes, both of which healed promptly after curettment without any bone destruction.

One case had a small ring sequestrum in the upper pin hole which healed promptly after its removal.

Three of the first four cases had some anterior bowing of the fragments due to the fact that a stabilizer was not used.

Two of these cases were further complicated by pressure necrosis of the skin over the upper fragment, following the use of a pressure pad to force the fragments into apposition.

One of these had delayed healing of the skin but not of the bone. The other had sufficient pressure to cause exposure of about two inches of the upper fragment with resulting dry necrosis, necessitating removal of the necrosed portion. This resulted in delayed union and slight anterior bowing, although good union had taken place in eight months.

The third case of bowing was easily corrected by application of a stabilizer after lengthening had been completed, and union took place without any delay or wound complications.

All of the later cases of tibial lengthening, on which the stabilizer was used, had no anterior bowing or separation of the fragments. In none of the cases was there any bowing or separation of the fibular fragments or delay of union.

The two cases of femoral lengthening need separate mention since a specially devised apparatus was used upon them. The apparatus used consists of a screw extension bar of aluminum alloy tubing, 18 inches long, upon which are mounted two reverse "T" shaped posts. One post is fixed to the end of the bar, the other moves along the bar by means of a screw device. Two pins are mounted on each of the "T" posts, one on either arm by means of swiveled chuck devices. These not only lock the pins in a rigid position when tightened, but allow for any slight variation in the alignment of the pins when apparatus is applied to them. The apparatus is so constructed that both of the "T" posts can be rotated with a key to correct any lateral bowing or displacement of the fragments, which may take place during the lengthening process.

The pins used are 3/16 inch stainless steel, six inches long with drill point. The distal one and one-fourth inches (1¼) of the pin is turned down 1/32 inch, leaving a shoulder. This prevents the pin's being forced too far through the bone.

The femoral lengthening operation is performed as follows:

A two-inch incision is made over the lateral aspect of the femur, just below the trochanter. The bone is exposed and the upper pin is drilled into the femur parallel to its transverse axis. A second incision is then made on the lateral aspect of the femur, just above the external condyle. The distal pin is then drilled into the lower end of femur. A double guide is then applied to the two pins which are in position, and the medial pins are then put in place, the upper and lower pins being about one and one-fourth inches (1¼) apart. The skin gaps between the upper and lower pins are then closed and temporary dressing applied. An incision about eight inches long is then made on the outer side of the femur and carried through the vastus lateralis muscle. The periosteum is split for about six inches and stripped from the bone. With a large bone saw the femur is split about 4 or 5 inches. The osteotomy is completed above posteriorly and below anteriorly with a blunt pointed osteotomy saw. The periosteum is then divided in a zig-zag manner in several places. The divided bone is then tied together with two ligatures of No. 2 chromic gut, and the wound is closed in three layers. Pin holes and lengthening wounds are dressed separately as in the tibial lengthenings.

The apparatus is then applied and firmly tightened to the pins. The leg is suspended in a modified Hodgkins splint with the knee extended.

Both of the femoral cases in this series were

lengthened approximately three inches. Because of the flexibility of the apparatus, very little lateral deviation of the fragments occurred and this was easily corrected. No anterior bowing took place during the process of lengthening. Some bowing did take place, however, in both cases, after removal of the apparatus, due to the fact that we failed to estimate the amount of bony union necessary to resist the strong bow string action of the hamstring muscles.

Results.—No major obstacle was encountered in any of the cases in this series. Of the seventeen cases treated, none was a failure. The minimum tibial lengthening was 1¾ inches, the maximum 2¾ inches. One femur was lengthened 2¾ inches and one 3¼ inches. All patients were up within three months in casts. Walking calipers were applied in four months. The bones were well regenerated in all cases in from 6-8 months.

CONCLUSIONS

From our experience with this limited series of cases we are of the opinion that:

Lengthening of the tibia and femur are procedures of great value in carefully selected cases.

Strict asepsis must be observed during the operation and the utmost care must be exercised during the lengthening process to prevent contamination of the wounds.

We believe drainage of the wounds is unnecessary.

Good apposition of fragments is essential to rapid union.

With the type of apparatus used frequent x-rays, which we believe are a factor in delaying union were not necessary.

Case 1.—V. V., a girl of 13, following poliomyelitis, had 2½ inches shortening.

Previous operations consisted of bilateral plantar fasciotomy June 29, 1927, and bilateral club foot stabilization June 29, 1927.

Leg lengthening data follow:

Tibial lengthening September 30, 1927.

Apparatus removed October 20, 1927.

Allowed up with cast and crutches January 14, 1928.

Brace applied February 1, 1928.

Almost complete regeneration March 10, 1928.

There were no complications. The legs are of equal length; all wounds are well healed.

Case 2.—J. T., a boy aged 12, following poliomyelitis, had 1¾ inches shortening.

Previous operations consisted of left plantar fasciotomy October 9, 1927, and left club foot stabilization October 9, 1934.

Leg lengthening data follow:

Tibial lengthening January 4, 1928.

Apparatus removed and plasters applied February 6, 1928.

Allowed up March 10, 1928.

Dismissed April 4, 1928.

Complete regeneration October 18, 1928.

There were no complications.

Case 3.—L. W., a girl of 13, following poliomylitis, had 2 inches shortening.

Previous operations were:

Right club foot stabilization April 29, 1927.

Right tendo achilles lengthening June 10, 1927.

Right anterior ankle block June 10, 1927.

Right tibial turning July 8, 1927.

Leg lengthening data follow:

Right tibial lengthening March 14, 1928.

Apparatus removed April 23, 1928.

Allowed up August 15, 1928.

Brace applied August 29, 1928.

There was delayed healing of pin holes from excessive granulation, necessitating curetting.

There was no observation following dismissal.

Case 4.—L. S., a boy aged 14, following poliomyelitis, had 2½ inches shortening.

Previous operations consisted of left foot stabilization August 13, 1934, and transplant of peroneals to os calcis August 24, 1934.

Leg lengthening data follow:

Left tibial lengthening May 30, 1928.

Apparatus removed and cast applied August 28, 1928.

Allowed up October, 1928.

Sequestrectomy November 7, 1928.

Delayed union of skin due to anterior bowing of fragments. Ring sequestrum removed October 7, 1928.

X-ray August 20, 1930, showed complete regeneration of tibia.

One-half inch shortening November 1, 1934.

Case 5.—E. H., a girl of 13, following poliomyelitis, had 2 inches shortening.

Previous operations consisted of right plantar fasciotomy May 24, 1929, and right club foot stabilization May 24, 1929.

Leg lengthening data follow:

Right tibial lengthening September 25, 1929.

Apparatus removed November 16, 1929.

Allowed up January 11, 1930.

There was irritation around pin holes.

Sinuses curetted December 27, 1929.

Wounds well healed. Tibia shows complete regeneration April 1, 1930.

Case 6.—R. E., a boy aged 12, following poliomyelitis, had 2 inches shortening.

Previous operations consisted of right foot stabilization April 23, 1926; right supracondylar osteotomy May 12,

1926, and right tendo achilles lengthening October 9, 1929.

Leg lengthening data follow:

Right tibial lengthening June 21, 1929.

Apparatus removed August 19, 1929.

Allowed up with crutches September 28, 1929.

Tibia well healed. Legs of equal length June 30, 1931.

There were no complications.

Case 7.—L. B., a girl aged 14, following poliomyelitis, had 1¾ inches shortening.

Previous operations consisted of stabilization of the left foot June 13, 1923, and left tendo achilles lengthening January 8, 1930.

Leg lengthening data follow:

Left tibial lengthening January 8, 1930.

Apparatus removed February 24, 1930.

Allowed up (in plaster cast) March 1, 1930.

Brace applied November 11, 1930.

Anterior sequestrectomy developed June 18, 1930, due to an anterior pressure pad.

The last observation was in February, 1932. The sinuses were all closed. The tibia was completely regenerated and the legs were of equal length.

Case 8.—L. B., a girl of 14, following poliomyelitis, had 2¾ inches shortening.

Previous operations consisted of stabilization of the right foot June 27, 1923, and right knee arthrodesis October 4, 1929.

Leg lengthening data follow:

Right tibial lengthening July 16, 1930.

Apparatus removed September 18, 1930.

Allowed up (in plaster) September 25, 1930.

Brace applied November 25, 1930.

There were no complications.

Observation January 1, 1931, shows complete regeneration of the tibia. The legs are of equal length.

Case 9.—A. P., a girl of 12, following poliomyelitis, had 1¾ inches shortening.

Left tendo achilles lengthening had been done October 7, 1931, and stabilization of the left foot October 7, 1931.

Leg lengthening data follow:

Left tibial lengthening June 8, 1931.

Apparatus removed August 10, 1931.

Allowed up (in plaster cast) September 19, 1931.

There were no complications.

Observation April 19, 1932, showed complete regeneration of the tibia, with no shortening.

Case 10.—W. M., a girl of 14, following poliomyelitis, had 2 inches shortening.

Previous operations consisted of left plantar fasciotomy April 15, 1927; left foot stabilization May 11, 1927; left peroneals to os calcis May 11, 1927; and left tendo achilles November 13, 1931.

Leg lengthening data follow:

Left leg lengthening August 7, 1931.

Apparatus removed September 16, 1931.

Allowed up (in plasters) October 10, 1931.

Brace applied December 14, 1931.

There were no complications. Observation in January, 1932, showed almost complete regeneration of the tibia. The legs were of equal length.

Case 11.—N. D., a girl of 12, following poliomyelitis, had 1½ inches shortening.

Previous operations consisted of stabilization of the right foot on April 10, 1929, and right tendo achilles lengthening January 8, 1932.

Leg lengthening data follow:

Right tibial lengthening August 12, 1931.

Apparatus removed October 12, 1931.

Allowed up (in plaster) October 17, 1931.

Brace applied November 12, 1931.

Complete regeneration of tibia October 25, 1932.

There were no complications.

Case 12.—B. S., a boy of 12, following poliomyelitis, had 2½ inches shortening.

Previous operation consisted of tarsal osteotomy to correct foot May 15, 1931.

Leg lengthening data follow:

Tibial lengthening August 12, 1931.

Apparatus removed October 12, 1931.

Allowed up (in plasters) October 17, 1931.

Brace applied November 12, 1931.

Tibia well regenerated February 4, 1932.

Last observation January 19, 1933.

There were no complications. There was no shortening. The tibia completely regenerated, and the legs were of equal length.

Case 13.—L. P., a girl of 13, following poliomyelitis, had 2 inches shortening.

Previous operations consisted of left supracondylar osteotomy June 17, 1932, and left tendo achilles lengthening August 10, 1932.

Leg lengthening data follow:

Left tibial lengthening August 10, 1932.

Apparatus removed September 26, 1932.

Allowed up (in plaster) October 22, 1932.

Brace applied December 19, 1932.

There were no complications and the tibia showed almost complete regeneration. Legs of equal length January 27, 1933.

Case 14.—P. J., a boy of 15, following poliomyelitis, had 1½ inches shortening.

Previous operations consisted of right foot stabilization with anterior ankle block December 19, 1930; arthrodesis knee January 7, 1931; and right tendo achilles lengthening January 6, 1933.

Leg lengthening data follow:

Right tibial lengthening June 29, 1932.

Apparatus removed September 5, 1932.

Allowed up October 22, 1932.

Dismissed in brace November 5, 1932.

Complete regeneration of tibia January 3, 1933.

There was delayed healing around the pin holes from excessive granulation, necessitating curetting. Eventually all wounds were well healed and the legs were of equal length.

Case 15.—H. L., a girl, aged 15, following poliomyelitis, had 4 inches shortening, practically all in the femur.

Previous operations consisted of left foot stabilization March 30, 1927; left hip stabilization April 22, 1927; left peroneals to os calcis May 20, 1927; and left supracondylar osteotomy July 6, 1932.

Leg lengthening data follow:

Left femoral lengthening January 18, 1933.

Apparatus removed, plaster applied June 17, 1933.

Leg lengthened 3¼ inches.

There were no complications. Observation in June, 1934, showed complete regeneration of the femur. Anterior bowing was very slight. There was one inch of actual shortening.

Case 16.—N. M., a girl of 14, following poliomyelitis, had 2¼ inches shortening, all in the femur.

Left pan arthrodesis had been done November 11, 1932.

Leg lengthening data follow:

Left femoral lengthening February 7, 1934.

Lengthening complete February 27, 1934.

Plaster applied March 12, 1934.

Allowed up June 25, 1934.

There were no complications. At the last observation in July, 1934, the femur was well regenerated. The legs were of equal length.

Case 17.—C. H., a girl of 12, following poliomyelitis, had 1¾ inches shortening.

At previous operations 1¾ inches actual lengthening had been accomplished.

Leg lengthening data follow:

Left tibial lengthening July 11, 1934.

Pins removed, cast applied September 12, 1934.

Allowed up October 13, 1934.

There were no complications. The legs were of equal length. She walked without external support. X-ray showed good bone regeneration.

DISCUSSION (*Abstract*)

Dr. Frank D. Dickson, Kansas City, Mo.—There is no longer any doubt as to the value of the leg lengthening procedures as begun by Abbott.

In our experience, there are two steps in the operation which seem very important. First, the lengthening of the heel tendon, since failure to have sufficient length results in equinus and often inversion of the foot. Even when paralysis of the calf muscles is present with a calcaneus, lengthening of the heel tendon is, we believe, necessary. Occasionally a posterior capsulorrhaphy is required. Second is the division of the deep fascia on the outer side of the leg, as it offers considerable resistance to lengthening if intact.

It seems essential in any apparatus for leg lengthening that a stabilizer be used; at least, it materially lessens the tendency to bowing. The apparatus used in our clinic employs four Kirschner wires instead of pins, but even with these bowing will occur, and we intend in the future to use in addition a stabilizer. Occasionally I have been able to control slight bowing by suspending the foot of the splint and permitting it to sag of its own weight so that slight backward bowing is produced.

Lengthening operations should be done with greatest care and only on those cases most carefully selected. I do not employ the procedure of lengthening the tibia and fibula in older patients, but feel that after the child has reached the tenth year it is better to lengthen the femur, or better still to wait until the child is from sixteen to seventeen years old and then shorten the opposite femur.

Dr. Stephenson (closing).—In our series of cases we thought it best to choose patients who were near the age of 13 or 14 in order to minimize shortening which may result from further growth. We feel that the procedure could be carried out on older persons if it were necessary.

In our series of femoral lengthenings we thought it best to lengthen the short femur due to the extreme amount of shortening. Dr. McBride mentioned lengthening the tibia on the foot that had a fusion of the ankle joint. We had two cases in this series who had previous fusions in the ankle joint. Legs were lengthened and no difficulty encountered.

PTERYGIUM OPERATION*

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Before the Section on Ophthalmology of the American Medical Association, at the Saratoga Springs, New York, session in 1902, I presented a brief paper on the nature and treatment of pterygia. The principles announced and the operative measures described have been so kindly received by my colleagues throughout the world that a word of profound gratitude cannot be withheld.

In order that I may more clearly emphasize the modifications in method which a more extended experience has developed, it would be well to refer again to the general principles involved, together with a brief description of the measures that are sufficient in the uncomplicated cases.

The details of the operation as described in the original paper in 1902 are as follows:

(1) The neck of the pterygium is grasped completely with strong but narrow fixation forceps.

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*Read in Clinical Session, Texas Ophthalmological and Otolaryngological Society, meeting conjointly with Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

(2) A Graefe knife is passed through the constriction and as close to the globe as possible, and then, with the cutting edge turned toward the cornea, every particle of the growth is shaved off smoothly from the cornea.

(3) The pterygium is still held with the fixation forceps, and with slender, straight scissors the conjunctiva and subconjunctival tissue are divided along the lower margin of the growth a distance of from one-fourth to one-half inch.

(4) While the pterygium is being held with the forceps, the body of the growth is separated from the sclera with any small, noncutting instrument.

(5) The conjunctiva lying below the oblique incision made with the scissors is now separated well from the sclera.

(6) A black silk thread is armed at each end with a small, curved needle. Both of these needles are carried through the apex of the pterygium from within outward and separated from each other by a sufficient amount of the growth to secure a firm hold.

(7) The needles are then carried downward beneath the loosened conjunctiva lying below the oblique incision made by the scissors. The needles, after passing in parallel directions beneath the loosened lower segment of the conjunctiva until they reach the region of the lower fornix, should then emerge from beneath the conjunctiva at a distance of about one-eighth inch from each other.

(8) Now, with the forceps, the loosened lower segment of conjunctiva is lifted, and traction gently exerted on the free ends of the threads which have emerged from below. The pterygium will glide beneath the loosened lower segment of the conjunctiva, and the threads may then be tightened and tied and the surplus portions of the thread cut off, leaving enough to facilitate the removal of the threads after proper union has occurred.

It is very important that no incision be made along the upper border of the pterygium, because it would gape and leave a denuded space when the downward traction is made on the pterygium. On the contrary, the elasticity of the conjunctiva is such that when this downward traction is exerted on the head of the pterygium, the conjunctiva becomes thinned out and smoothly applied to the sclera corresponding to the former site of the body of the growth, and the margin of the conjunctiva coincides accurately with the sclerocorneal junction. Thus, when the operation is completed and the speculum removed, no stitch is seen, because it is hidden by the lower lid; the only denuded area is the cornea. The former site of the body of the pterygium is covered by a thin and comparatively non-vascular conjunctiva, what blood vessels remain are directed downward and not horizontally, and hence do not tend to encroach again on the cornea. In fact, the whole vascular activity is concentrated beneath the lower lid, where it is not only removed from view, but also protected from the irritating influences of dust and exposure, and the process of atrophy naturally and surely follows. In the meantime, the corneal wound heals quickly and the thin conjunctival tissue becomes closely adherent to the sclera in the palpebral opening. After a few days the single stitch can be removed and the old pterygium will be found firmly adherent to the sclera and hidden beneath the loosened lower segment of the conjunctiva. If the head of the growth is very large, so that it covers something like a third of the cornea, and if the body is also thick and fleshy, it may be best to dissect away the head from the cornea with a sharp knife and then remove with scissors a part of the head and a part of the subconjunctival portion of the body of the growth, and then deal with the rest of the pterygium, according to the method already described.

In the case of pterygia involving the nasal aspect of the globe, if the growth is transplanted downward and a liberating incision is found necessary, this incision should be made upward in a direction approximately that of a vertical tangent to the limbus instead of in the direction approximately of a horizontal secant to the limbus. If the pterygium is to be transplanted upward, the same general principles will apply. If the pterygium involves the temporal aspect of the globe, the same general principles will govern whether the growth should be transplanted upward or downward.

In ordinary cases, complete anesthesia can easily be obtained by instillations of 5 per cent solution of cocaine, or a derivative of butyl amino propane ("butyn.") But when the growth is very thick and fleshy, the so-called pterygium crassum, I invariably apply a few crystals of cocaine hydrochloride directly to the head and neck of the pterygium, and this procedure insures absolutely perfect anesthesia, so that there will be no delay in the successful performance of every detail of importance.

With reference to agents employed in advance to control hemorrhage at the time of operation, I will say that the judicious use of epinephrine chloride can do no harm in any case; but I reserve this for the more vascular forms of pterygia. Of course, it may be said that the action of epinephrine chloride will render less distinct the outlines of the growth and thus interfere with the accuracy of the procedure; and while this in a measure may be true, still I am convinced that if the operator possesses good sight and a true hand, he will always be able to remove completely every vestige of the growth. On the

contrary, there are some elements of value in epinephrine chloride in addition to its power of controlling hemorrhage; for when the pterygium is very broad and fleshy, it so largely conceals the knife transfixing the growth that one may not be able to gauge properly the depth of the corneal incision. Now, the action of epinephrine will increase the transparency of the growth and enable one more definitely to determine the situation of the advancing knife.

I have employed a special pterygium knife, which is essentially a narrow, short-bladed, double-edged keratome, placed at an angle of 135 degrees with the axis of the handle; in reality, a very narrow, angular keratome. This instrument enables the operator, by standing at the head of the patient, to introduce the knife with equal facility on either side of either eye, and also to cut in any desired direction without changing the relation of patient and surgeon. It is essential that the knife be very narrow, so that it can transfix the neck of the growth without first cutting laterally to any marked extent. Then, when the point has well emerged from beneath the neck of the pterygium, the knife can be made to shave off every particle of the growth from its corneal attachment.

In this connection, I cannot emphasize too strongly the absolute necessity of the complete removal of the growth from the cornea. I have repeatedly demonstrated that, in a given pterygium, if a portion of the head is radically removed and a portion is imperfectly removed, the tendency to recurrence is manifested in far greater degree in that part corresponding to the incomplete removal. Now, the pathology of pterygia clearly proves that the growth, if fully developed, involves not only the epithelial layer and Bowman's membrane, but also the superficial lamellae of the corneal stroma. Moreover, as stated above, experience has abundantly shown that any procedure which fails to include every vestige of this abnormal corneal involvement must also fail to secure the nearest approach to perfect result with the minimum tendency toward a recurrence. Hence it is that all forms of divulsion and ablation by means of dull instruments must fall short of the highest efficiency, because they necessarily fail to reach the basal layers of the corneal invasion, and they leave a surface better adapted to the return of the growth. I do not mean to say that excellent results have not been attained by divulsion, for I have many times successfully employed this method myself; but I am thoroughly convinced that such a procedure, while it may yield excellent results in some localities, cannot stand the test of a comparison under atmospheric conditions that strongly favor the development of pterygia. In other words, in some localities almost any method will succeed, while in others a successful result will exact more careful attention to every detail of thoroughness and every element of safety. Since a radical removal of the head is a matter of great importance, it is essential to the highest efficiency that the pterygium knife should be as sharp as possible, both in its point and in its cutting edge. In fact, I have found it useful to guard with as much care of the keenness of a pterygium blade as I would that of a cataract knife.

In very broad and fleshy pterygia, the usual plan of transfixion and subsequent ablation, as described above, must be modified in accordance with the area involved. Suppose, for instance, a pterygium covers one-third of the cornea with a dense vascular mass, and one transfixes the neck with an instrument having a straight cutting edge like a Graefe cataract knife; when the knife advances over the cornea from the neck to the apex, its area of cleavage, or track or path, is not the surface of a sphere with the normal corneal curvature at all times equally distant from the membrane of Descemet, but its path is a single plane, cutting off a segment of a sphere whose periphery involves only the most superficial layers of the cornea, while its center may involve the entire thickness of the cornea. In such cases the head of the growth must be dissected away from the cornea thoroughly but carefully, and the success of this procedure will depend in large measure on the sharpness of the instrument employed and the facility with which it may be made to change its direction in accordance with the curvature of the cornea, thus permitting the operator gently to insinuate the blade of the knife between the affected superficial lamellae and the normal deeper lamellae.

It is important also to remember that this process of complete removal must extend to the neck of the growth as it passes over the limbus. The separation of the body of the growth from the underlying sclera, to which it is always loosely attached, except in cases in which operation has been performed unsuccessfully, may be accomplished by a variety of instruments. I generally employ for this purpose the point of the closed pterygium scissors, which is useful also in undermining the conjunctiva above the pterygium, and in making the subsequent oblique incision along the lower border of the pter-

ygium and in undermining the conjunctiva below this oblique incision, and also in removing, when necessary, any portion of the growth or of the vascular subjunctival tissue beneath it. It sometimes happens that the head of the body of the growth may be so large and bulky that it is unnecessary, for the smooth performance of the operation, to preserve every portion of this growth in transplanting, and so I am in the habit of cutting away in such cases the superfluous tissue. But it is essential that this cutting away process be confined to the horny portion of the head, the lower margin of the body, and the vascular subconjunctival tissue, always preserving intact the superior margins of the pterygium, in order that we may be able to utilize to the fullest extent the elasticity of the unbroken conjunctiva above the growth in covering smoothly the denuded area of the sclera when downward traction is made on the head of the pterygium.

In order that the pterygium may easily and freely glide downward when traction is exerted on it, we must be careful to undermine the conjunctiva thoroughly above the growth, and especially must we ascertain that all conjunctival fibers in the immediate vicinity of the neck shall be freed from their attachments at the limbus. The exact position of the oblique incision along the lower border of the growth would necessarily vary with the breadth of the growth, and in no case should this incision encroach on the pterygium either in its body or at its neck; and no harm would result, but greater safety be secured, by allowing a margin of 1 mm. between the lower border of the growth and this oblique incision.

THE SUTURES

I have tried all forms of suture material. That which has been most satisfactory has been fine, black silk thread, although absorbable suture, if sufficiently fine, will give perfect results. I formerly allowed the stitch to remain four days, but I have found that sufficient adhesions will frequently occur within two or three days, and this is a matter of some moment to patients who live at a distance and who would prefer to remain away from home under the direct care of the oculist only until the stitch has been removed.

The direction of the two threads as they pass almost parallel beneath the loosened lower segment of the conjunctiva may vary somewhat according to the elasticity of the conjunctiva and the scleral area to be covered by the attenuated conjunctiva from above the growth. Generally, they will emerge near each other in the region of the lower fornix under the center of the cornea, but these positions may be modified slightly, according to the denuded area to be covered and the degree of traction that is exerted on the threads. The essential object to be obtained is so to direct the threads and so to regulate their tension that the normal conjunctiva above the growth shall be made to curve accurately around the denuded limbus without encroaching on the cornea or without leaving any portion of the sclera uncovered. If the calculations have not been perfectly accurate and one finds some overlapping of the cornea, the needles should either be introduced again or the overlapping conjunctiva should be carefully trimmed away along the line of the limbus.

After the tying of the threads has arrested the insignificant oozing that may sometimes occur, it is well carefully to inspect the field and especially to ascertain whether every vestige of the growth has been removed from the cornea. If it has not been thoroughly removed, the corneal wound will not present a transparent stroma, but will show a certain degree of vascularity or opacity which will indicate the necessity of further attention. Then we can again resort to a perfectly sharp pterygium knife and shave off every particle of the pathologic tissue, leaving a smooth surface over a transparent cornea.

Since a pterygium may require removal in the later decades of life, when the patient may present some tendency toward glaucoma, I have sometimes felt that it was prudent to instill a few drops of pilocarpin solution after the operation. My attention was first directed to this by a patient who came to me absolutely blind from acute glaucoma that had supervened immediately on a rather protracted operation for extensive pterygium in the hands of a competent and experienced confrere.

CONCERNING THE TREATMENT OF RECURRENT CASES

As to the methods of treatment, I would say that I have, at different times, tried out thoroughly every imaginable method of destroying the growth: by chemical means, by electricity, and by the various forms of surgical intervention. All forms of destruction by chemical and electrical agents lead to severe reaction, which, in turn, produces a correspondingly large amount of scar tissue, the evil genius in ophthalmic surgery.

All medical and hygienic treatment will fail to cause the disappearance of the growth in any

case, but may retard or arrest its progress and relieve the hyperemia. An error in refraction is seldom an important factor, but correcting lenses not only assist the vision, but offer mechanical protection against the impact of particles of dust.

Operative measures offer the only means of complete and permanent cure, and even then the prognosis will depend upon many conditions that can be recognized in advance and will enable us to give a more dependable prognosis. The prospect for a satisfactory result will be more unfavorable the more frequently the patient has been unsuccessfully operated upon. Each recurrence renders the situation more difficult because of the increased amount of scar tissue resulting. In fact, many of these cases that have often recurred no longer present the problems of an uncomplicated pterygium, but rather those of a definite symblepharon, or even a keloid formation, limiting the ocular movements and producing diplopia. I have received many inquiries from my ophthalmological friends as to the best procedures under such circumstances, and I regret that the answer is not more encouraging.

It is of primary importance to remember that the ordinary methods for simple pterygium will fail in these cases. If the cicatricial contractions are not marked, it will be admissible to dissect up the conjunctiva carefully from the subjacent scar tissue, taking care to preserve intact all of the available mucous membrane.

Then the scar tissue itself should be thoroughly removed from the sclera to which it is attached, thus liberating the eyeball and giving it mobility. (I have seen some patients in whom the internal rectus had been unfortunately sacrificed in an effort to radically remove the growth, so it is important to proceed with the utmost care in this dissection.) Then, the loosened conjunctiva should be drawn over the denuded sclera and securely fastened to it with sutures, so that no sliding of the conjunctiva will be possible to interfere with prompt and firm adhesion between the conjunctiva and sclera. The essential objects to be attained are the preservation of all available conjunctiva, the elimination of all scar tissue, and the effective union of the conjunctiva to the denuded scleral surface.

These measures may be employed when the amount of mucous membrane is adequate, but when there has been too much destruction of the conjunctival membrane it will then be necessary to resort to some kind of epithelial grafting. The mucous membrane of the lips and other parts of the oral cavity are too vascular and red to yield a good cosmetic result, but the healthy abundant bulbar conjunctiva of another person, or epithelial grafts from the inner surface of the arm, can be successfully used in covering the scleral defect. The grafts should be of such size and shape that one margin coincides with the limbus, while the other margins can be tucked under the overlapping edges of the loosened conjunctiva. The epithelial grafts from the arm are whiter than is desirable at first, but they tend gradually to acquire the proper color. Finally, there are some cases in which there has been so much destruction of tissue and so much scar formation that we might just as well refrain from any operative procedure.

COMPLICATIONS

(1) *Ulceration of Cornea.*—It would seem that in those cases involving extensive areas of the cornea and penetrating to the deeper layers of the stroma beneath Bowman's membrane sometimes an active ulceration would supervene. And yet, I have not seen any case in which this condition obtained. When the invasion has been very great, I have tried to minimize the risk by careful preparation of the eye before the operation, by the substitution of other local anesthetics for cocaine, by the free use of hot fomentations to keep up the nutrition, and by moderate use of miotics to prevent such stasis in the lymph flow through the cornea as would follow from the development of an increase in the intraocular pressure.

(2) *Glaucoma.*—I have observed varying degrees of intraocular hypertension, amounting in some instances to acute glaucoma, as a secondary complication to protracted operative procedures under cocaine, especially in aged people. To guard against this unfortunate possibility, I have followed the same general plan already outlined to prevent ulceration of the denuded corneal area, with perfectly satisfactory results.

(3) *Uveal Inflammations.*—As a complication of corneal inflammations, we should not be surprised to find sometimes an involvement of the iris and ciliary body, but this condition I have observed only occasionally, and then it was of moderate intensity. It was encountered mainly in the days when we were employing various electrical and chemical measures to remove the

growth or to prevent a recurrence. In other words, the tendency to iritic involvement seemed to be proportionate to the corneal reaction, and this was more pronounced when we depended upon agents whose field of action we could not perfectly control.

(4) *Suppuration in the Subconjunctival Cellular Tissue.*—A few times I have seen a mild suppurative inflammation of the cellular tissue covering the anterior segments of the globe, but I have never seen a postocular cellulitis develop, or even a general tenonitis. The suppuration has been always in the region of the stitch, which was evidently the avenue of infection. In such cases we have always immediately removed the suture and a free incision was made through all of the infiltrated tissues down to the sclera. After a few days the process subsided and the recovery proceeded in the usual way.

PROGNOSIS

The prognosis is the thing of chief concern to the patient, and any study is profitable that will help us to foretell with reasonable accuracy the outcome of any surgical procedure.

In general terms, I should say that any pterygium that is rather rapidly advancing would call for a guarded prognosis, because the underlying conditions leading to this advance are still active, and if not modified, would continue to exert an influence after the operation just as they did in producing the growth in the first place. The more vascular forms, the so-called pterygium crassum, show a marked tendency to recur, and call for a period of judicious aftercare, while the thin stationary, nonvascular types, the pterygium tenue, would yield a satisfactory result and under almost any kind of rational treatment. And yet, it is just in this vascular type that an effective operation can accomplish the most good, and it is also in this type that a faulty operation and ill-advised postoperative measures will lay the foundation for recurrence and all the attending regrets. Therefore, it is wise to study each individual case with the purpose of eliminating, as far as possible, all of the contributing causes at the time the operation is performed, because it must be remembered that no operation can leave the eye in a better condition than it was before the growth began, and if the pterygium can originate in an otherwise normal eye, it can recur after any operation, should the eye be continually subjected to the same exciting factors.

TECHNIC AND USES OF SUSPENSION LARYNGOSCOPY*

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Your indulgence is asked for reviving an old subject, one developed and presented by masters of our profession. I refer to suspension laryngoscopy devised and presented by Killian, perfected and popularized in this country by Lynch. To Lynch we are indebted for the standardized technic of suspension laryngoscopy as used today.

In my work the suspension laryngoscope plays an important part and excels by far any other method of direct laryngeal examination. Suspension laryngoscopy is primarily intended for the direct examination of the larynx and as a means of approach to intralaryngeal surgery. It may also be used in cases of supraglottic tumors, new growths occurring in the hypopharynx, base of the tongue and mouth of the esophagus. I use the suspension laryngoscope by choice for the introduction of the bronchoscope when doing an endoscopy, removing it as Jackson removes his laryngoscope when the tube is once introduced into the trachea. For the simple introduction of the bronchoscope the suspension crane is never used. The larynx is quickly exposed, the suspension laryngoscope is supported by the left hand while the bronscoscope is introduced under direct vision. The removal of the suspension laryngoscope is quickly accomplished to permit that freedom of motion necessary in doing endoscopy. I am cognizant of the fact that the bronchoscope may be introduced directly or by the aid of other laryngoscopes. The suspension laryngoscope helps me not only for the introduction of the bronchoscope but it has served me excellently in the examination of the larynx in those cases presenting histories of inspired foreign bodies. It has been my privilege to remove under direct vision safety pins and other foreign bodies embedded in the larynx and subglottic region since this method permits the free use of both hands, a condition necessarily advantageous, particularly when dealing with sharp-pointed foreign bodies.

The application and use of suspension laryngoscopy are extremely simple and can be acquired with but little practice. Local or general anes-

*Read in Clinical Session, Texas Ophthalmological and Otolaryngological Society, meeting conjointly with Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

thesia may be used. My preference for the latter is responsible for the fact that the majority of my cases receive a combination of ethylene, oxygen and ether. Those cases requiring cauterization or diathermy coagulation are given chloroform anesthesia. This latter anesthetic was adopted only after a most tragic experience and is considered the only safe general anesthetic to be used in the presence of a spark. In local anesthesia I use sodium isoethylamyl barbiturate followed by morphia and an application of 10 per cent cocaine to the base of the tongue, epiglottis, hypopharynx and in edentulous mouths to the upper gums. Before introducing the supsension laryngoscope I place two sterile towels under the head of the patient. The lower one serves to cover the table while the upper one is wrapped around the head of the patient covering the eyes which are protected by a piece of wet cotton placed over them. Complete relaxation, particularly in adults, is necessary when doing suspension laryngoscopy. A side mouth gag is used and the suspension spatula is introduced along the base of the tongue, avoiding any trauma to the posterior pharyngeal wall. The laryngeal surface of the epiglottis is engaged with the tip of the spatula. If the selection of the spatula has been accurate the tooth plates of the suspension unit will fit snugly against the lead plate previously fitted to the upper front teeth. This latter plate is made of sheet lead and serves to protect bad dentures as well as the enamel surface of the teeth.

The lead is easily moulded to the shape of the individual teeth and helps to distribute the pressure which is exerted against the upper jaw. Slight traction is made on the suspension unit while the thumb screw opens the gag fixing the tongue in the mid-line. When this is accomplished the hook is engaged to the gallows and the patient's head, so to speak, is suspended. This is, however, a misnomer, for in reality the head of the patient is seldom, if ever, lifted completely off the table. Just enough upward pull is exerted by the crane to produce extension of the head sufficient to convert the curved tract from the teeth to the vocal cords into a straight line with the mouth open. In order to do this it is necessary to obtain sufficient relaxation of the muscles of the neck, plus the application of a certain amount of power and force. This power must be applied in a manner that will not jeopardize the welfare of the patient and must be safe in every way.

In teaching laryngoscopy I find that the greatest difficulty experienced by my students is the fixing of the tongue in the median line and the exposure of the anterior commissure. Failure to do either one of these would probably result in a poor exposure of the larynx and a possible traumatism of the glosso-palatine muscle. Supraglottic tumors or lesions on the base of the tongue, lateral pharyngeal wall or pyriform fossa may require the insertion of the spatula to one side of the tongue for a better view of the field. Exposure of the anterior commissure depends greatly upon the anatomical structure of the patient. A long thin neck with poor muscular development is the ideal for suspension demonstration, while a short thick neck invariably presents difficulty in exposing the anterior commissure. By turning the handle on the crane which brings the cross bar toward the operator it is possible to angulate the tip of the spatula upwards thereby giving an eighth to a quarter of an inch more space which at times is sufficient to show the anterior commissure.

Frequently a small child is brought into the office with laryngeal involvement or a pediatrician will request the examination of the larynx of a youngster for continued hoarseness or suspected diphtheria. In my experience it has been occasionally very difficult to obtain the desired information by the use of the laryngeal mirror. I find myself resorting more and more to laryngeal suspension for a deliberate and accurate diagnosis in these youngsters. This type of examination is done in the office and takes only a few minutes. The little patient is wrapped in a sheet and placed on a table. My office nurse holds the head in the extended position while I introduce a mouth gag and quickly follow this by inserting the suspension laryngoscope into the mouth. An excellent view is obtained of the larynx. In these cases there is no necessity for the use of the suspension crane; the left hand simply supports the Lynch unit without any exertion. For the examination of infants I have a special spatula, a size smaller than that supplied with the apparatus.

The excellent view obtained by means of the suspension laryngoscope plus the decided advantage of having both hands free for diagnostic or surgical interference should be of sufficient importance to recommend its universal use. The ability to retract or palpate a lesion occurring in the hypopharynx or larynx is of considerable clinical importance. Deliberate examination of the subglottic area by means of the tilting mirror, or the examination of the under surface of the

vocal cord by retraction can best be accomplished by suspension laryngoscopy. Frequently I encounter a lesion which taxes my diagnostic ability. An area of tissue is questionable and the mode of procedure depends upon the macroscopic diagnosis. When such a condition presents itself I find great comfort in requesting the presence of the pathologist at the time of the operation, so that he too may sit at the head of the table and with a headmirror deliberately examine, palpate and render an opinion on the case in question.

The dissection and cauterization of intrinsic carcinoma occurring on the vocal cords is a well known procedure. Lynch reported a large number of cases successfully operated upon. I wish to add eight cases that have gone from two to four years without recurrence. I do not refer to these cases as cured, but prefer to speak of them as well without recurrence. The technic used in these cases consists of exposing the larynx by means of the suspension laryngoscope. The affected cord is engaged by means of the Lynch side grasping forceps and traction is made toward the opposite cord. With a sharp laryngeal knife the cord is dissected out. A wooden tongue depressor fitted onto a special metal holder is introduced edgewise between the cords, flattened out and slight pressure made downward. This opens the glottis to its maximum, fixing the cords in one position and giving ample space in which to apply diathermy coagulation to the base of the lesion without fear of injury to the opposite cord. Very little reaction is experienced by these cases and in due course of time, nature re-manufactures a pseudo-cord which serves excellently in speaking. Early recurrences at the original site in these cases are subjected to further cauterization or coagulation of the lesion. I have seen a number of recurrences requiring several coagulations before they could be referred to as well without recurrence. Those whose recurrences are extensive immediately become candidates for laryngectomies.

Suspension laryngoscopy to me is indispensable in the treatment of multiple papillomata of the larynx, particularly in those cases where there is an abundant growth of papilloma requiring an extensive removal. Supraglottic tumors, depending upon their attachment, are best handled by using a short broad spatula, frequently one which does not engage the epiglottis. Many cases of laryngeal stenosis present real problems to the laryngologists. I find the use of suspension laryngoscopy a decided help when dealing with such cases. Under suspension the surgeon may work through the larynx and the assistant may help by rendering aid through the tracheotomy wound or vice versa.

In this day and time when rigid economy is being preached and practiced, any instrument which serves a multiplicity of purposes is of decided value and the suspension laryngoscope does what all other laryngoscopes are intended to do. In addition it gives me a better direct exposure of the larynx than it has been possible for me to obtain with any other instrument I have seen. By permitting the free use of both hands surgery of the hypopharynx and larynx has not only been simplified but the possibilities of intralaryngeal surgery have been broadened considerably. Those who saw the motion pictures of the larynx which I recently presented at the Academy meeting in Chicago are thoroughly convinced of the excellent exposures obtained of the normal and pathological larynx. Suspension laryngoscopy is so indispensable and valuable to me that I feel it deserves and should enjoy a more universal use.

THE MANAGEMENT OF BLOOD STREAM INFECTION FOLLOWING MASTOIDITIS*

ILLUSTRATED WITH LANTERN SLIDES

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The diagnosis, medical and surgical treatment of sinus thrombosis constitutes one of the most brilliant and illuminating chapters in the entire realm of medicine and surgery. If all cases presented the classical textbook symptoms, the subject would be of little interest, but more often each case presents some variation which makes the diagnosis and treatment a difficult matter. The purpose of this paper is not to attempt any original suggestion, but rather for an exchange of opinions with the view of evaluating procedures which are in dispute. Some of the unsettled problems are as follows:

(1) In cases where thrombosis of the sigmoid

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sinus is complete, should the jugular vein be ligated?

(2) Where there is complete thrombosis of the sinus, should all of the clot be removed in order to secure free bleeding?

(3) What surgical procedure should be used in dealing with the vein: (a) ligation, (b) ligation and division, or (c) ligation and resection?

(4) What should be done to the facial vein?

(5) What should be done to the external jugular and emissary veins?

(6) Do the vein and sinus function after ligation?

(7) What place, if any, has chemotherapy in the medical treatment?

INCIDENCE

Sinus thrombosis is the most common of the intracranial complications of the middle ear and mastoid infections. Statistics vary, but the average incidence is between 5 and 10 per cent for both acute and chronic mastoiditis. The acute exacerbations of chronic mastoiditis are prone to produce intracranial complications.

DIAGNOSIS

In the presence of known mastoid infection before or after operation, if there is a sudden rise in the temperature once or twice in twenty-four hours, with or without chills and coincident with a rise in the white blood count, it is an almost definite indication that infection has entered the blood stream. This is the most significant single symptom. It is, of course, assumed that other systemic conditions have been excluded by a thorough physical examination. Seydell,[1] in a brilliant monograph on this subject, has pointed out that rather frequently fever is the only symptom present in an infection of the lateral sinus. He classified the condition as follows:

(1) The pyemic type, which is characterized by pyemic fever. The temperature may range from 96 to 106° F.

(2) The septic form, in which the temperature remains high, the pulse is fast, of poor quality, and remains so during remissions.

(3) Most frequently, septicopyemic, where the temperature may be continual, strongly remittent or intermittent.

Chills do not occur as often as the textbooks indicate. Blau[2] gathered from the literature one hundred cases of sinus thrombosis, none of which presented chills. Takatabake[3] carefully studied fifty-one cases and only six had chills. In our experience chills occurred in one-third of our cases. Blood cultures should be taken in suspected cases at twenty-four - hour intervals if negative. Carmack[6] has shown that if cultures are taken before the chill and rise in temperature, a greater number of positive results will be obtained. Positive cultures should be obtained in 75 per cent of all cases. A positive or negative blood culture should never be the sole determining factor in the surgical management of a suspected case of sinus thrombosis. One should always realize that a complete thrombosis of the sinus with negative culture is possible and a positive culture without a sinus thrombosis.

Study of the Blood.—In every suspected case a complete blood count should be made daily. It tells us whether the disease process is going or coming. Ordinarily the leukocyte count is parallel with the severity of the infection. A Schilling hemogram is of much greater value in determining the presence of a severe systemic infection. Eosinophils disappear from the blood in severe infections and their reappearance is the earliest favorable sign. If other complications have not occurred, there should be a rapid increase in the red cells and hemoglobin after the infection is eliminated from the blood stream. A study of the hemoglobin and red cells is an indication of the need of blood transfusions.

Study of the Bacteriology.—Bacteriological studies should begin when the myringotomy is performed, also when the mastoid is opened. The urine should be centrifuged and stained for bacteria. Wanamaker[4] has been able to find streptococci in the stained smears in sinus thrombosis before the appearance of the organism in the blood. The hemolytic streptococcus is the organism most frequently found.

Changes in the eye grounds occur in a small percentage of cases. When present they vary from a slight fullness of the retinal veins to a choked disc. This condition subsides following thorough drainage of the mastoid. The Tobey-Ayer test is of definite value where there is complete obstruction of the sigmoid or jugular bulb on one side. In performing the test, the patient should be relaxed and the pressure should be made on the jugular vein, continuously, for one minute. There is always a sharp rise in cerebrospinal pressure in the normal individual when one jugular is compressed and this returns to normal within thirty or forty seconds if the other side is patent. It is the sustained

pressure or increasing pressure after thirty to sixty seconds that is diagnostic. Spinal puncture should be done in all suspected cases of intracranial involvement, withdrawing just enough fluid for diagnostic purposes and noting carefully the increase, if any, in the pressure.[6]

TREATMENT

The treatment may be divided into medical and surgical with equal emphasis. Good results have been reported from non-surgical treatments, but the method of choice is to make intelligent use of both medical and surgical treatment. The most effective medical therapy is the transfusion of whole blood. This should be given while we are waiting for definite diagnostic symptoms, sometimes during the operation and during the period of convalescence. The hemoglobin concentration, the red blood count and the general well-being of the patient are the indications for the time to give the blood transfusions. If the hemoglobin content is sixty or below, we give transfusions every forty-eight hours. We give from 100 to 150 c. c. The importance of giving whole blood at frequent intervals cannot be too strongly emphasized. We are dealing with a serious condition and a delay of a few hours, not days, often is the difference between success and failure. The donor and recipient should be carefully matched before each transfusion. Both may change. The use of antistreptococcus serum and mercurochrome have not been of value in our cases. Neoarsphenamine, however, has been of definite value and we use it in all cases, giving a small dosage intravenously not more than once a week. Intravenous saline and glucose is often used with excellent results. The surgical treatment consists of elimination of the primary focus of infection, prevention of dissemination of the infection from areas with thrombophlebitis, drainage of the vein involved and the treatment of any metastasis. The first step calls for a thorough mastoid operation simple or radical, depending upon the indications. Since it is not always possible to differentiate by symptoms and laboratory tests a sinus clot from a bacteremia, one is justified in performing a mastoid operation and freely exposing the sigmoid sinus. Slightly more than the width should be exposed for inspection and compression if it is decided upon later. The appearance of the bony sinus plate is not to be relied upon. Oftentimes we find a sinus thrombosed or abscessed, yet the bony plate was apparently sound. The mastoid emissary should be exposed if present. If there is only slight evidence of phlebitis present, no other surgery is done. The wound is left open and particular care should be used in packing the wound. There is little likelihood that the sinus will become infected if the packing is not placed against it. If the septic symptoms are more pronounced and there is definite evidence of thrombosis, the jugular should be ligated and the lateral sinus opened and drained. In cases of complete thrombosis, the questions arise: (1) Should the clot be curetted until free bleeding occurs from each end? (2) Should the jugular be ligated before opening the sinus? There is a difference of opinion among the best known otologists of the country on these questions. Our own policy is to ligate the vein before opening the lateral sinus. We do not attempt to remove all of the thrombus from the sinus in order to secure free bleeding. The thrombus is nature's attempt to stop the infection. When the thrombus is removed and free bleeding occurs, gauze plugs have to be used. Gauze plugs are objectionable. (1) One has a foreign body in an infected field adjacent to the dura with the possibility of producing trauma to the brain and possibly meningitis or an abscess. (2) There is the problem of removal. It is usually five or six days before these plugs can be removed.[6]

It is our policy to open the sinus for about one inch and drain by use of a soft rubber catheter. Never use curettes or any metallic instruments. The opening in the sinus is packed with iodoform gauze to prevent sudden bleeding if there is breaking down of the thrombus. The packing can be removed in two or three days instead of the usual five or six days and this lessens irritation of the brain and general toxemia.

PROGNOSIS

The prognosis depends upon the thoroughness of the primary mastoid operation, the timely and careful administration of whole blood transfusion, the thorough obliteration of foci of infection from areas of phlebitis in accessory veins, the emissary and external jugular and the surgical skill employed in opening and draining the sinus. In this connection the time required for ligating the vein should be considered. If for any reason the operation requires more than thirty minutes, the patient should have intravenous glucose and saline or blood transfusion while on the table. If this is not done, it is best to do the two procedures in stages rather than exhaust the patient's resources by a prolonged anesthesia and operation.

In the patients who had positive blood cultures

prior to operation and negative afterwards and a corresponding decrease in leukocyte and polynuclear cells, and in whom the Schilling hemogram shows a constant turn to the right, one can feel assured of a good prognosis. Patients with metastasis limited to the extremities usually offer a favorable prognosis.

Organisms may be found in the blood for months after the operation and exacerbations with fatal termination occur fairly often. The necessity of observing patients carefully for months following blood stream infection cannot be too strongly emphasized. Avoidance of work, worry, exposure to cold and general supportive and hygienic measures should be rigidly enforced. Death may result from emboli in the lungs or elsewhere, meningitis, pneumonia and general toxemia.

SUMMARY

Lateral sinus thrombosis is the most common of the intracranial complications following middle ear and mastoid infection. It occurs most frequently in men and in both acute and chronic ear infections.

Sinus thrombosis may arise from other sources than the mastoid cells, namely: (1) Infection from the scalp through the mastoid vein, occipital diploic and posterior temporal diploic veins and through the superior longitudinal and the cavernous sinus. (2) The bulb may become involved by direct extension from the middle ear or from the labyrinth through the internal auditory vein and the inferior petrosal sinus. We may also have a thrombosis from infections in the neck. While these avenues of infection exist and must be recognized, the majority of all cases of involvement of the lateral sinus result directly from an extension of the mastoid infection with necrosis of the surrounding bone. Usually a perisinuous accumulation of pus forms with bacterial invasion of the vessel wall. It is possible in practically all cases of sigmoid involvement to find a necrotic fistula in the bone extending from the mastoid cells to the sinus. Blood stream infections from thrombophlebitis of the sigmoid may usually be classed as the result of delayed or neglected mastoid drainage. [1]A generalized infection following a suppurative process in the middle ear and mastoid is so often associated with sinus thrombophlebitis that the terms otogenous pyemia and sinus thrombosis are frequently regarded as synonymous. Haymann[5] has shown by his experiments that bacteria may pass into the sinus wall and enter the circulation without the presence of a thrombus in the sinus. It is this type of infection that responds to medical treatment. It unquestionably explains why some writers report favorable results in sinus thrombosis without surgical interference with the sigmoid sinus and internal jugular vein. Emphasis should be placed upon the importance of daily blood studies in every suspected case. When the hemoglobin concentration and the erythrocyte count continue to fall, whole blood transfusion should be given. The responsibility for the management of these cases rests entirely with the otologist and he must carefully and diligently supervise the laboratory work, the blood transfusions as well as the surgical treatment. Any case which has not been ligated at the time of the mastoid drainage should be ligated within seventy-two hours unless definite improvement is observed. The same treatment of the vein applies to the cases of complete thrombosis of the sinus. It is not necessary or advisable to remove all of the clot from the sinus in order to secure free bleeding.

Free drainage[6] through an incision about one inch long with careful removal of the clot only at the point of the incision will permit drainage from either end, if there is breaking down of the thrombus. Section of the vein should rarely, if ever, be done and that only when it contains a clot. If only ligation has been done under perfect asepsis the wound should be closed. The jugular vein should be ligated above the facial vein. If the jugular vein is ligated below, then the facial vein should be ligated.

[7]The external jugular vein communicates directly with the internal jugular vein. Cases have been reported where recovery was delayed until the external jugular was ligated. The mastoid emissary connects the posterior auricular and the sigmoid sinus. It often passes through pneumatized bone and may become involved primarily. Examination of the patency of the emissary is a valuable diagnostic aid in suspected thrombophlebitis of the sigmoid. It is not patent in about 25 per cent of cases. Involvement of the emissary may be by continuity of tissue or by extension of the thrombus from the sigmoid and should be considered in all cases of lateral sinus thrombosis.

There have been sufficient experimental work and clinical observations by experienced otologists to establish clearly the fact that the circulation of blood recurs following ligation of the jugular vein and drainage of the sigmoid sinus. The only chemotherapeutic agent which has proven of value in our hands is neoarsphena-

mine. Antistreptococcus serum and bacteriocides have been used without benefit. Reduction in mortality rate depends upon:

(1) Prompt and thorough mastoid drainage. (2) Careful inspection and palpations of the sinus wall in all suspected cases. (3) Early ligation and drainage of the sinus and obliteration of areas of thrombophlebitis. (4) Whole blood transfusions as indicated by the hemoglobin concentration, the erythrocyte count and the well-being of the patient.

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NEWER KNOWLEDGE OF VITAMINS*

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The subject that has been assigned to me for this Symposium is one that presents tremendous difficulties in its presentation. When one stops to consider the fact that the first important book on vitamins appeared in 1913, and that prior to this time the knowledge of the subject was but fragmentary and totally incomplete, and then stops to realize how much we know at the present time about these important substances, the vitamins, he can secure an impression of the immense amount of material which may be, strictly speaking, considered as being "newer knowledge." Even the volume by Funk which appeared in 1922 is but rudimentary and elementary in the light of present day knowledge, with tremendous gaps in the information concerning the role that the vitamins play in disease and in health. Furthermore, were I to attempt to review the literature of the last two years it would be a mountain of a task virtually insurmountable. During the year 1932, for example, there were 332 papers published which had to do with vitamin D alone and a few less than a thousand papers on vitamins as a whole. In 1933 the flood of vitamin papers was still uncontrolled and I am sure that when the summary of 1934 makes its appearance it will be found that this year has been even more prolific in the production of papers concerning vitamins than any previous year. However, the work of the reviewer of these papers is very much simplified due to the fact that in each of the last three years L. J. Harris, of the Nutritional Laboratory of the University of Cambridge, England, has abstracted critically and reviewed the more important contributions that have appeared each year and has published this in the *Annual Review of Biochemistry*. In the preparation of this paper I have drawn freely and liberally on these reviews of Harris.

In an attempt to bring out the newer knowledge of vitamins I trust it is obvious from what I have just said why a paper of this length would be inadequate. Fortunately, I am limited somewhat in the breadth of my field, as much of the newer literature has to do with the chemistry of the vitamins, and their isolation, as well as clinical aspects of the dietary deficiencies.

Although it has been only in the last few years that the importance of the role of vitamins in clinical medicine has been greatly appreciated, and, although it has only been in the last decade or two that intensive work has been done on this subject, it must not be forgotten that scurvy was recognized by the ancients, that nearly 200 years ago James Lind appreciated the relationship of the disease scurvy to dietary deficiency. Even more antique is beriberi, which, according to McCollum, was known to the Chinese more than 2,500 years before the birth of Christ. Descending to more modern times pellagra was adequately described exactly 200 years ago. It really was not, however, until the fundamental contributions of Eijkmann that the relationship of the symptoms of beriberi to a diet lacking some essential factor was demonstrated.

VITAMIN A

It has been shown definitely in the last two years that this important vitamin is present in certain yellow plants which contain the provitamin A, carotene. The distribution of this substance in the past year has been intensively studied. Halibut liver oil is the richest known source of vitamin A and D. Milk is an important source of this vitamin and there is comparatively little seasonal variation in the vitamin A content of milk, nor is there much difference in the milk of various breeds of cows, nor from

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various types of animal life. In plant life the vitamin appears in yellow rooted vegetables and in green leafed vegetables. The richest plant spring of vitamin A is the apricot.

Deficiency of vitamin A, in this country at least, is uncommon, but in parts of Asia, for example, lack of it is believed to be a chief cause of early blindness and Carillo says in the Yucatan one child out of every five suffers from xerophthalmia. The condition of xerophthalmia, or nyctalopia, is well-known, but perhaps it is not as well appreciated that there are certain other expressions of vitamin A deficiency which only recently have been recognized. Thus changes take place in the teeth or a peculiar type of dermatitis may be the result of vitamin A lack, and sterility occurs in the experimental animal. Scott states that vitamin A is probably of greater importance in the prevention of sterility than is vitamin G. The effect on the vaginal mucosa, comparable to the cornification that occurs in the eye, prevents the proper reception of the spermatozoa. Vitamin A is transmitted to the infant through the mother's milk, but often the nursing parent suffers from lack of or exhaustion of this vitamin.

Particularly interesting has been the work of the last few years, stimulated largely by Mellenby, on vitamin A as having an anti-infective action. Indicative of the amount of work that has been done on vitamin A as well as other vitamins in relation to the response to infection, is a review of the subject by Robinson in *Medicine*, which in scope is nearly a hundred pages long. She very adequately and fully discussed the evidence *pro* and *con* and is somewhat guarded in her conclusions. However, it might be said that there are many publications in the last year which deal with vitamin A therapy. It has been tried on a large scale at various places and in many conditions. The evidence forthcoming would tend to show that if there is any tendency to infection, which is doubtful, as a result of vitamin A inadequacy in conditions, for example such as puerperal sepsis, otitis media, scarlet fever or pneumonia, it would depend largely upon the keratinization of the protecting epithelium and not upon any inherent value of vitamin A in stimulating immunologic processes in the body. Furthermore, it is certainly appreciated that in this country at least vitamin A shortage is an extremely exceptional condition.

One last word on vitamin A deficiency. A new method which is comparatively simple has been devised by Jeans and Zentmire to determine moderate degrees of vitamin A want when not sufficiently extreme to produce clinical manifestations. This is a light-eye test which seems to be of value. These authors found that of 213 children 149 were regarded as normal, whereas 45 indicated poor sensitivity to light. These latter children were kept in the hospital and given cod liver oil and in an average period of twelve days the light sensitivity had returned. They find that about 21 per cent of the children examined had a certain degree of vitamin A deficiency.

VITAMIN B

A recent paper by Balfour and Talpade illustrates the fact that vitamin B deficiency is remarkably prevalent in Asia, and that nearly all pregnant women in Southern India are in a state of avitaminosis. Premature births are three times as common in this section of India as in the North. In Japan, 17,000 deaths are believed to be due to beriberi every year. In this country, because vitamin B is found only in fair amounts in milk, it is quite possible that sub-nutrition may be a common expression of lack of this vitamin in the suckling, proven by the improvement that occurs in weight and height when extra vitamin B is added to the diet.

The chemical studies that have been made with the various fractions of vitamin B will not be mentioned here. Of some interest, however, are the papers which have dealt with the occurrence of vitamin B in various foods. Thus animal foodstuffs contain more B_1 than do the vegetables, but not as much as do cereals. Interesting to note that dried brewer's yeast which is commonly employed in the treatment of pellagra may vary in its vitamin B_2 content as much as 10 per cent (Seidell). It is also disconcerting to hear from the Ohio Agricultural Experiment Station that vitamin B may be destroyed to as high as 25 per cent by the process of pasteurization.

Harris says that the "outstanding event of the year has been the identification of vitamin B_2 (or a major component) as the naturally occurring water-soluble pigment" first described in 1879 and now broadly spoken of as flavine. However, this has to do with the chemistry of the vitamins and will not be discussed further. Of particular interest is the report of Hogan and Richardson, who have been able to produce skin lesions which others have been unable to obtain in the experimental animal when the anti-der-

matic vitamin is withdrawn from the diet. The diet is irradiated, which may explain the result obtained, as vitamin B_2, a flavine pigment, is destroyed by the action of light. This work has not been completely confirmed, however.

In regard to pellagra, the disease is extremely common. In the South the mortality figures confirm this. It is rather interesting to note that a not inconsiderable number of cases have been discovered recently in Denmark and in England. All the papers dealing with pellagra have not by any means been unanimous in asserting that this condition is dependent upon a vitamin deficiency. Spies, for example, recounts his experiments in which pellagrins improved even when given a vitamin-poor diet.

The relation of vitamin B to anemia is an extremely lively and argumentative question. A considerable number of recent papers tend to uphold the theory that macrocytic anemias of several types are dependent upon vitamin B_2 inadequacy, notably the work of Castle and his coworkers. Vitamin B_2, however, does not replace liver in the treatment of pernicious anemia and furthermore Brand, West and Stucky have found an antianemic factor that cures pernicious anemia which, although associated with vitamin B_2 crudely, when further purification is carried on, is quite free from it. Likewise vitamin B_2 from egg white does not affect the pernicious anemia patient. These papers do not substantiate Castle's idea that vitamin B_2 is the extrinsic factor in pernicious anemia. However, there can be no quarrel with this author, in that it is closely associated with it.

A very interesting development in regard to vitamin B_2 has been the experimental demonstration that glossitis occurs in animals kept on a low vitamin B_2 diet. These experiments would seem to show that the glossitis of sprue and pernicious anemia may be brought about by absence of some part of the vitamin B complex. One last interesting clinical observation is the possible effect that vitamin B_2 lack may have of bringing about some disturbance of iron metabolism. Sargant has reported that irrespective of the anemia, the nervous expressions of subacute combined sclerosis will greatly improve by enormous doses of iron, which work has been confirmed by Biggam and Ghalioungui in relieving the nervous lesions of pellagra by iron therapy more rapidly and simply than by the usual dietary measures.

Vitamin B_3 has been described with an action supposed to be growth stimulating. More recent work would tend to show that this factor depends upon giving a more adequate supply of B_1 so that the existence of vitamin B_3, at least the alleged existence, is in doubt.

Vitamin B_4 is also being mentioned in literature at the present time as a fraction of vitamin B which has an antiparalytic effect. That vitamin B_4 exists is probably true, but that it has been isolated is a question which still must be considered *sub judice*.

Another interesting and thought-provoking paper of recent vintage is that by Strauss, who says that many of the expressions of vitamin and dietary deficiency diseases may be dependent upon gastro-intestinal disturbances more than on inadequate diets. He discusses the part that the gastro-intestinal tract plays in some of the conditioning deficiencies of certain of the vitamin B complexes.

VITAMIN C

Here again the most important contribution in the last few years to the study of this vitamin has been its chemical identification as ascorbic acid. This has been a development of practically the last year.

Another chemical observation of considerable moment is the estimation of vitamin C in human tissue. It is found to be greatest in the adrenal, brain, pancreas and other tissues. The average value in the adrenal was 0.55 mg. per gram as contrasted with 0.04 mg. for heart tissue. This is of some moment on account of the discussion that has taken place concerning the physiologic action of vitamin C and more particulary in its relation to the adrenal. The idea seems to be that a liberal amount of vitamin C is necessary for the normal working of this organ rather than that it is stored there for usage of the rest of the tissues, as the liver stores up vitamin A. Vitamin C lack is so common in this country that the nursing infant is started on orange juice or tomato juice long before he is weaned. Even if the baby is breast-fed the vitamin probably should be given to him, but even more necessary is the giving to the small baby of adequate amounts of vitamin C when he is artificially fed. The vitamin is easily destroyed in the milk according to Kon, and even in fresh milk it may be lacking. Exposure to light seems to be the main cause of this inactivation which goes to show the importance of nature's method of giving milk of the mother to the child. Another interesting development in the study of vitamin C in the last couple of years has been the demonstration by Smith and McConkey that guinea pigs deprived of vitamin

C often develop gastric ulcers; an important observation in view of the tendency at the present time to feed the gastric ulcer patient on a diet lacking fruit juices.

A large scale experiment was tried out in Chicago by Hanke, who gave large doses of orange juice to the children in Chicago, which he contends prevented gingivitis and certain common dental disorders.

The capillary resistance test has been a method of diagnosing subclinical scurvy supposed to be present when petechial spots occur below or at the site of the tourniquet. Greene has presented a paper in which he concludes that the capillary resistance test is not an accurate nor adequate method of determining an insufficient vitamin C dietary.

VITAMIN D

Harris calls attention to the prevalence of rickets. A few years ago 87 per cent of the London school children had some degree of this abnormality. It is said that in urban communities a third of the white and a half of the negro children have some of the expressions of rickets. This fact is so well known that, as with orange juice and tomato juice, the pediatrician begins feeding his small infant patient viosterol or cod liver oil while still a nursling.

Vitamin D in its most available form, viosterol, is derived from irradiated ergosterol, which in turn is a sterol which is a solidified alcohol. Irradiation of food will supply the needed vitamin. The evidence that irradiated food is popular is seen in the enormous amount of money that has been received by the University of Wisconsin from the Steenbock patent which makes possible the irradiating of foods. Vitamin D is required by the pregnant woman during lactation in from three to five times the normal amount. Unfortunately, as pointed out in a current editorial of the *Journal of the American Medical Association*, milk is anything but a good source of vitamin D and even if the lactating mother drinks milk she probably does not get enough to supply her child.

Some of the recent work that has to do with vitamin D concerns the prophylactic and curative doses of viosterol, the former being 1,500 units and the latter 3,000 units. Toxic ill effects may occur with as little as 10,000 units. Considerable interest has been displayed in the last several years concerning the possibility of viosterol overdosage. Reed recounts the symptoms that occur as a result of too large doses of viosterol in patients who were given doses of from 3,000 to 2,760,000 international units daily. In this group of patients those that had symptoms attributable to viosterol suffered from nausea, vomiting and diarrhea. Loss of appetite was quite common and there was a tendency to gain weight, although in the experimental animal there is an increase in the metabolic rate. Muscular weakness and lassitude, dizziness and dis-coordination were also noted. There is a general impression that viosterol has a distinct aphrodisiac effect. In this group of 50 men studied by Reed who were questioned as to "libido, frequency of intercourse and general interest in sex life" it was found that viosterol had no aphrodisiac effect and in the group of women only one admitted any stimulation of libido. Some of the women reported some disturbances in the menstrual flow. Reed says that there is little need of worrying about the administration of viosterol in amounts up to 150,000 international units daily. King and Hull found that massive doses of viosterol given to chickens produced a marked hypercalcemia and ultimate death.

Vitamin D is another one of the vitamins that may have an effect in one way or the other on dental caries. It would seem from a contemporaneous publication that the important feature in the diet is deficiency in phosphate rather than lack of vitamin D.

The physiology of vitamin D action has been quite extensively studied. One of the hypotheses of its action is that it stimulates the parathyroid, but a recent paper by Harris and Parks shows that there is a fundamental difference between the action of vitamin D and parathyroid. There is a considerable number of papers on this question which is largely one of chemical interest.

It may be of some interest to know that a not inconsiderable number of papers has appeared dealing with methods to increase the antirachitic activity of cow's milk. Such efforts have to do with feeding the cows viosterol, irradiating their food or even irradiating the animals themselves. One last word in regard to cod liver oil and that is that the Norwegian cod gives a liver oil which is 50 to 100 per cent richer in vitamin D than does the cod that is caught off the New Foundland banks.

VITAMIN E

This particular vitamin is of greater interest to the gynecologist than it is to the internist. It is a vitamin which is known as the anti-sterility vitamin and which, when withheld from the ex-

perimental animal, occasions abortion. There are several interesting observations in regard to this vitamin in the last few years aside from the gynecologic aspect which apparently is of very minor clinical importance. One of these observations is by Hill and Burdett, who say that "royal jelly" is able to convert the larva of a working bee into a queen. Another observation is that sterility occurs not only in the female but also in the male. Certain histologic changes occur in the testes when there is lack of vitamin E. These structural changes are preceded by fundamental physiologic disturbances. If the lesions in the testes develop, the gonads are not restored to the normal even by a diet high in vitamin E consumed over a long period of time. Dryerre says that abortion is common in Scotland amongst the sheep. Thirty-six animals were treated with a concentrate which was made up of vitamin E, A and D. Only one of these sheep aborted, whereas this happened in 20 per cent of 650 controls.

VITAMIN G

This vitamin has been discussed in conjunction with vitamin B. The present day tendency is to disregard the term G and speak of this vitamin as B_2 or B_2G or even $G(B_2)$.

VITAMIN F

Particulary intriguing in regard to the subject of vitamins is the recent work published by Evans, *et al*, who fed animals upon diets which lacked the so-called essential fatty acids, but with all other dietary essentials. They not only fed to their animals a complete diet, but they actually supplemented this diet with increased amounts of vitamins A, E, D and carotene. Their observations tend to show that successful gestation upon the fat-free diet was impossible, whereas the addition of the essential unsaturated fatty acids which are hesitantly spoken of as vitamin F showed a marked improvement in the birth mechanism. On this insufficient unsaturated fatty acid diet lactation was also disturbed. The male animal on this diet invariably became sterile, but small amounts of the essential fatty acids cured as well as prevented this sterility. Here, then, we have a new vitamin added to those which are already recognized. This vitamin does not seem to have any relationship to vitamin E.

Other vitamin-like elements have been described in recent literature, but the work seems too immature at the present time to comment upon.

SUMMARY

I have attempted to bring out sketchily some of the more recent work in vitaminology. I should like to accentuate the growing importance of the study of vitamins which is vital to the health and to the propagation of the human race. I believe that it is at the present time the most lively subject in medicine and parenthetically one about which the laity probably knows more than of any other phase of medicine, incomplete as the knowledge may be.

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Discussion follows paper of Dr. Youmans, page 847.

CHEMISTRY OF VITAMINS*

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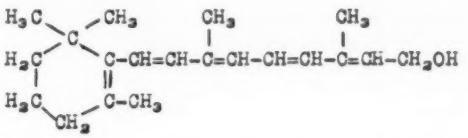
Great advances have been made in the study of vitamin chemistry during the last few years. Five of the vitamins, namely, A, B_1, B_2, C and D, have been isolated in an apparently pure form and their empirical formulae reasonably well determined. The provitamin of both A and D is known and C has been synthesized in the laboratory. A complete discussion of the chemistry of all the vitamins is, of course, impossible in the time at my disposal. Consequently I expect to discuss only the most important ones, and even in those cases the discussion must naturally be brief and to some extent incomplete.

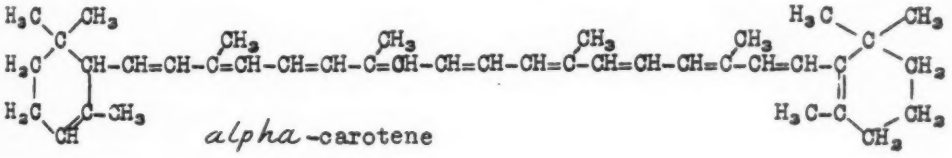
VITAMIN A

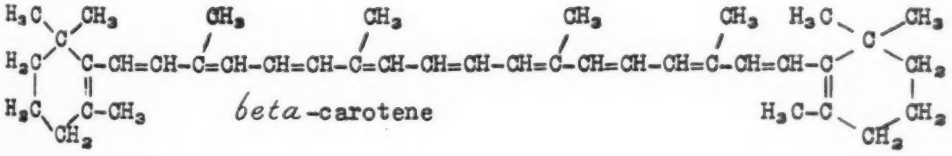
The chemical constitution of vitamin A, as determined by Karrer and his associates,[1] is as follows:

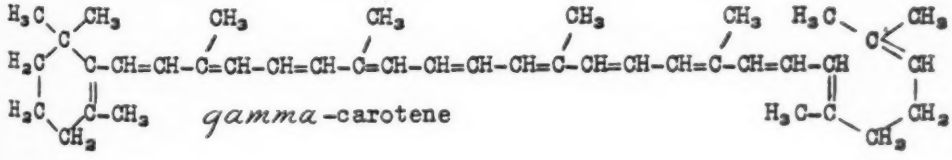


It will be seen that it is a primary alcohol and contains four double bonds in the side chain and one in the ring. This marked degree of unsaturation probably accounts for its relative instability. Provitamin A is the naturally occurring plant pigment carotene. Carotene occurs in nature usually, if not always, as a mixture of two or more isomeric forms, known as *alpha, beta, gamma,* and possibly still another one. The chemical composition of these isomers differ slightly, but all have at least one *beta*-ionone ring, a grouping which seems necessary for vitamin activity.

It will be seen that *beta*-carotene is symmetrical and contains two *beta*-ionone rings. Two molecules of vitamin A could be formed from it by breaking it down at the middle double bond with the formation of a primary alcohol at the terminal carbon atom. This might arise from the taking on of two molecules of water. *Alpha* and *gamma* carotenes are not symmetrical and







*Read in Section on Medicine, Symposium on Deficiency Diseases, Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

*From the Department of Biochemistry, Medical College of Virginia, Richmond, Virginia.

contain only one *beta*-ionone ring. They consequently would theoretically form only one molecule of vitamin A on being broken down. This is well supported by experimental evidence. The vitamin activity of *beta*-carotene, in small concentrations, is double that of *alpha*-carotene.[2] Furthermore, if moderate amounts of *alpha*- and *beta*-carotene are fed to vitamin A free rats, the livers from the *beta*-carotene fed rats will have approximately double the amount of vitamin A present in the livers from the *alpha*-carotene fed rats.[3]

Nothing definite is known regarding the chemistry of vitamin A action in the animal body. It is undoubtedly involved in maintaining the integrity of the epithelial system.

VITAMIN B COMPLEX

The vitamin originally known as vitamin B has been divided into several fractions. The separate identity of B_1, B_2 or G and B_4 has been definitely established, while considerable evidence has accumulated indicating the presence of other fractions also. However, only the first three will be discussed in this paper.

Vitamin B_1.—This vitamin is commonly known as the antineuritic vitamin. Crystalline preparations of it have been isolated by several investigators, but some doubt still exists regarding its chemical composition. It is known to be an organic base and to form salts with acids. Windaus, Tschesche and Ruhkopf[4] consider its empirical formula to be $C_{12}H_{16}N_4OS$. Ohdake[5] and Jansen, Wibaut, Hubers and Wiardi[6] give formulae which differ slightly from the above.

Considerable evidence has been brought forward in the last few years indicating that this vitamin is in some way associated with carbohydrate metabolism, perhaps in some oxidation mechanism. Glucose tolerance, as measured by the blood sugar curve, is less in B_1 avitaminosis than in normal animals. Furthermore, the accumulation of lactic acid in the body, after exercise, is much greater in cases of beriberi than in normal individuals, indicating that there is some disturbance in the mechanism for the conversion of lactic acid to glycogen or its oxidation to carbon dioxide and water. The bradycardia resulting from B_1 avitaminosis in rats is known to be due to lactic acid accumulation and may be cured by its removal.[7] *In vitro* experiments show that the oxygen uptake of minced brain tissue from B_1 avitaminous pigeons, when mixed with lactic acid, is less than is taken up by the minced brain of normal pigeons; also, addition of vitamin B_1 concentrate increases the oxygen consumption.[8] However, the actual mechanism by which the vitamin influences carbohydrate metabolism is unknown. Harris[9] suggests that it may enter into the mechanism of an oxidative system, such as a dehydrogenase system, in a role corresponding to that of a coenzyme. In other words, it activates the removal of hydrogen from the substrate by the dehydrogenase.

Vitamin B_2 *or* G.—One of the outstanding developments in vitamin chemistry during the last year or so was the identification of vitamin B_2, or a major component of it, as the naturally occurring water soluble yellow pigment lactochrome[10] (now renamed lactoflavine). It was first found in milk as long ago as 1879. This lactoflavine may be identical with ovoflavine from eggs. They show the same vitamin activity. The elementary composition of lactoflavine is given by Kuhn and his associates[10] as $C_{17}H_{20}N_4O_6$ or $C_{16}H_{20}N_4O_6$. Flavine is relatively inactive when fed alone, but when fed to rats in conjunction with vitamins B_1 and B_4 good growth takes place even from the administration of only 5 gamma per rat per day.

Flavine dissolves in water to give a yellow solution with an intense green fluorescence. Strong reducing agents convert it into the leuko or colorless form, but the original material can be readily regenerated by simply shaking it with air. Exposure to visible light, especially the blue-violet portion, bleaches it and renders it inactive. Gyorgy, Kuhn and Wagner-Jauregg[11] discuss the probable relationship of flavine to the so-called "yellow oxidation enzyme" of Warburg and Christian.[12] Apparently this enzyme consists of flavine in combination with a colloidal carrier. This enzyme acts as a carrier catalyst. It takes up hydrogen from the substrate and is itself changed to the colorless or leuko form. This leuko form is in turn oxidized by oxygen to the original enzyme with the formation of hydrogen peroxide. The reactions involved may be written somewhat as follows:

(1)	RH Substrate	+	B Oxidized form of enzyme	=	R Oxidized substrate	+	BH Reduced or colorless form of enzyme	
(2)	2BH Colorless form of enzyme	+	O_2	=	B Oxidized form of enzyme	+	H_2O_2	

The regenerated or oxidized form of the enzyme may now act on another molecule of substrate and the process is repeated.

Since this enzyme is probably necessary for the animal body and since the animal organism is apparently incapable of synthetizing its major constituent, "flavine or vitamin B_2," the necessity for the presence of this vitamin in animal food becomes evident.

Whether flavine is identical with the pellagra-preventive factor or whether it is one of a number of growth-promoting substances present in a complex and composite B_2 remains to be seen. In this connection Harris[9] writes:

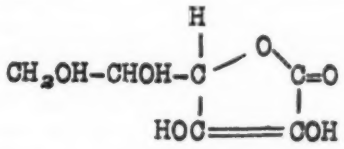
"Thus it is known irradiation of a diet confers dermatitis-producing properties on it, and it would seem reasonable to link this with the fact that such treatment does in fact rapidly bleach and destroy the flavine. Again the seasonal incidence of or photosensitization to pellagra might, I would suggest, be explained to some extent by the destruction of the flavine by the action of bright sunlight either on the natural food materials or, *in vivo*, on subjects exposed to it."

Vitamin B_4.—This vitamin is heat and alkali labile, like B_1. Its composition is unknown, but there is considerable evidence that it is in some way associated with the organic base adenine. It has been suggested by Winterstein and Schön that both vitamins B_1 and B_4 are necessary for the prevention of beriberi, while B_2 and B_4 are necessary for the prevention of pellagra.

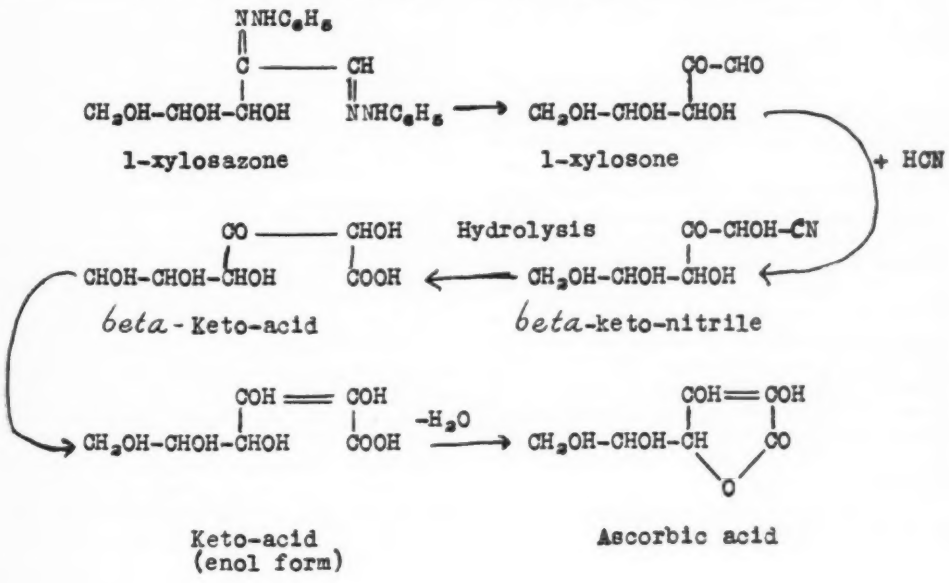
VITAMIN C

Although vitamin C is the most readily destroyed of all the vitamins, it was the first to be isolated in a pure form and the only one so far synthesized in the laboratory. In 1928 Szent-Gyorgyi isolated from the adrenal cortex and later from oranges and cabbages a crystalline substance, having the empirical formula $C_6H_8O_6$, to which the name hexuronic acid was given. After its identity with vitamin C was established by King and Waugh[14] and Szent-Gyorgyi and Svirbely[15] in 1932, its name was changed to ascorbic acid.

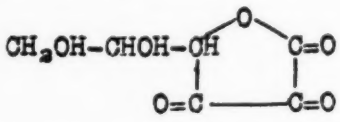
The constitution of ascorbic acid has been the subject of a number of investigations. The structural formula which is generally accepted is as follows:



In 1932, Reichstein and his associates[16] synthesized the vitamin. Starting from l-xylose, they prepared first the osazone and from this the osone. Treatment of the osone with hydrocyanic acid gave the nitrile, and the hydrolysis of the latter gave ascorbic acid. The synthesis may be represented as follows:



The most characteristic chemical property of vitamin C is its extraordinary power of reduction. In acid solution it may be oxidized with the loss of 2 hydrogens to a substance having the probable formula[1]:



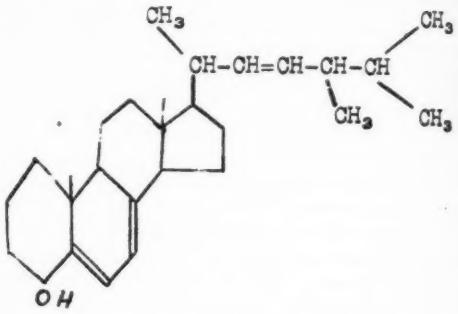
This oxidation product, known as dehydroascorbic acid, may be readily reduced to its mother form by hydrogen sulfide. It possesses a vitamin activity comparable to that of ascorbic acid itself.

The chemical mechanism of vitamin C action in the animal body is unknown. Because of the ease with which it undergoes reversible oxidation it seems reasonable that it may function as an oxygen carrier in the cell. That this is probably so is indicated by experiments of von Euler and Klussman[17] and Harrison,[18] who found that slices of tissue from scorbutic animals have a low oxygen uptake, and furthermore the oxygen uptake could be increased by the addition of ascorbic acid. Söderström and Törnblom[19] found the oxygen consumption in scurvy is lower than normal. Another interesting physiological function of vitamin C, as shown by Karrer,[1] is its activating action on the cellular proteolytic ferment cathepsin.

VITAMIN D

This vitamin is considered to be the same as calciferol, the vitamin active substance produced by the action of ultra-violet light on ergosterol. However, considerable evidence has accumulated during the last couple of years indicating that ergosterol may not be the only provitamin D. A good review of the literature is given in a paper by Wadell.[20] Five well characterized isomerides of ergosterol have been isolated from the products of ergosterol irradiation. They are and probably occur in the following order: ergosterol, lumisterol, tachysterol, calciferol, suprasterol I, suprasterol II. Of these crystalline substances only calciferol possesses antirhachitic properties. The last two are products of over radiation.

The ergosterol formula which seems to offer the most satisfactory explanation of the known reactions of ergosterol is given by Rosenheim and King[21] as follows:



The structural change which takes place in the molecule in its conversion to calciferol or vitamin D is unknown. It is known that the position of the double bond in the side chain is unimportant for the saturation of this double bond still leaves the molecule capable of activation by irradiation. Apparently the essential change is in the cyclic part of the molecule. It is of considerable interest that the vitamin has a phenanthrene nucleus, a structure common with several other physiologically highly active substances such as the female sex hormone and the carcinogenic hydrocarbon. Furthermore, ergosterol, calciferol and especially neo-ergosterol possess estrogenic activity; also some actively estrogenic substances are definitely carcinogenic.[22]

Physiology of Vitamin D Action.—No definite agreement has so far been reached regarding the physiological action of vitamin D. It is claimed by some that it functions by stimulating the parathyroid glands, thus increasing blood calcium and phosphorus and inducing calcification. Others are of the opinion that it acts primarily on the intestinal tract. Harris and Parks[23] have demonstrated that vitamin D diminishes calcium and/or phosphorus excretion in the feces. This reduction in fecal excretion might arise either from increased absorption or decreased excretion by way of the gut. However, the experimental results of Harris and his associates[24] and of Morgan and his coworkers[25] definitely indicate that vitamin D reduces the intestinal excretion of calcium. They consequently conclude that the primary function of vitamin D is to reduce this re-excretion and not to stimulate the parathyroid.

VITAMIN E

The constitution of vitamin E, the anti-sterility vitamin, is still unknown. It is thermostable and is not inactivated by hydrogenation or saponification processes. However, bromine, acetic anhydride and long exposure to ultra-violet light inactivates it. Although it occurs in nature associated with fatty materials, rancid fats very readily inactivate it. Nothing definite is known regarding the mechanism through which this vitamin brings about its physiological action.

SUMMARY

We thus see that in the last few years great strides have been made in determining the chemical structure of vitamins as well as the mechanism of their physiological actions. Little is known regarding the mechanism of vitamin A, B_4 and E action. Vitamins B_1, B_2 and C seem to be involved in oxidation processes. Vitamin B_1 possibly acts as a co-enzyme to a dehydrogenase, while B_2 is an important constituent of the "yellow oxidation ferment" of Warburg and Christian. Vitamin C, through its ability to undergo reversible oxidation, probably acts as an oxygen carrier in cellular metabolism. Vitamin D influences calcium and phosphorus metabolism, possibly acting primarily by diminishing intestinal calcium excretion.

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Discussion follows paper of Dr. Youmans, page 847.

SOME CLINICAL ASPECTS OF DIETARY DEFICIENCIES*

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The present day concept of disease resulting from a dietary deficiency is relatively new. Undoubtedly it was the discovery of the vitamins and their importance to health which stimulated this unique idea of a negative as contrasted with a positive etiology. Since then the concept has broadened to include not only all the vitamins and certain elemental substances, as iodine and calcium, normally required by the body in relatively small amounts, but even protein which constitutes a large part of the normal diet. In the case of the latter, finer distinctions may occur as when a deficiency concerns an important amino acid.

Much of the advance in our knowledge of the disease-producing possibilities of dietary inadequacies has been the result of animal experimentation. Not only have such studies clarified our knowledge of the more extreme and better known deficiencies, but they have suggested the possibility of other disease states. These include in addition to those which may arise from the lack of newly discovered specific substances, latent or subclinical forms of the better known deficiency diseases. The latter result from a slight or moderate but chronic shortage of the necessary food factors, either relative or absolute.

However, when one attempts to transfer the results of these animal experiments to clinical medicine, numerous difficulties and failures are encountered. These failures are the result of many causes which will be discussed later in this paper. At present I wish to illustrate some of

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the difficulties which occur without in any sense attempting to cover the entire field of the deficiency diseases.

For the past five years my associates and I have been studying at the Vanderbilt University Hospital a group of patients who have subsisted on an inadequate diet. For the most part these diets are not the result of the present economic disturbance, but represent the long established dietary of a considerable number of people in the community. Many of the patients have had no disease other than that which could be attributed to dietary deficiency and the study included some who were not considered ill. In addition, as would be expected, deficiency states are found complicating many other illnesses. The diets are inadequate in many respects. Deficiencies have been found in total calories, in protein, animal protein especially, and in vitamins A, B and C. In some cases there appear to be shortages in calcium and phosphorus, and perhaps in iron and iodine. In most individuals the diet is deficient in more than one and often in many of these factors.

Such diets as these might be expected to result in any one or more of several deficiency diseases, for example, beriberi, pellagra, anemia, nutritional edema and scurvy. Pellagra does occur among this group. Other patients present symptoms and signs which have been interpreted as due to dietary deficiencies and are suggestive of at least latent forms of one or another of these diseases. In many instances, however, the signs and symptoms are difficult or impossible to classify according to the specific deficiency diseases recognized at present and discrepancies occur which are hard to reconcile with knowledge gained from experimental procedures.

A common syndrome which these patients present is that of an uncomplicated edema not related to cardiac or renal disease. In previous papers I have expressed the belief that the edema itself is due primarily to a lowering of the serum proteins. This belief is still held and most, if not all, of the discrepancies between the serum protein level and the presence or absence of edema can be explained on the basis of secondary factors such as exercise, posture, temperature, tissue pressure, *et cetera*. However, the exact mechanism responsible for the lowered serum protein is not completely understood. That an inadequate intake of protein is, in a large measure, responsible, cannot be doubted. In nearly all of these patients the amount of dietary protein, particularly animal protein, is small and the scarcity is often exaggerated by the small caloric value of the diet. Nevertheless, an adequate intake of protein, while sometimes effective, does not always cause a rise in the serum protein and in many cases the response is very slow. In this respect many of these patients differ from experimental animals in which a normal or high protein diet causes a prompt rise in the serum protein when the latter has been depressed by plasmaphoreses or low protein feeding.[1] A similar prompt response to a normal or high protein diet is usual in cases of epidemic nutritional edema[2] and in many sporadic cases provided there is no interference with absorption. In our cases the impression is gained that restoration of the serum protein to normal levels is relatively rapid in the acute, but much slower in the chronic cases. In some instances complications, such as diarrhea, which may be due to chronic deficiencies, have undoubtedly interfered with proper nutrition. In others it is possible that difficulties in absorption or utilization are present which are not apparent and which may, themselves, be the result of dietary inadequacies. However, no definite evidence of decreased absorption has been obtained in those cases in which studies have been made.

Although an inadequate intake of protein has existed in most of these cases, occasionally an edema with lowered serum protein has been found in individuals whose diet was apparently adequate in respect to protein. This suggests that other deficiencies, that of vitamins for example, may influence the serum protein levels in these individuals. Elsom[3] has recently reported a case of edema and hypoproteinemia in which the administration of vitamin B caused a prompt rise in the proteins of the blood. In our experience, however, the administration of potent vitamin B preparations has had no significant effect on the serum protein levels. It is, of course, impossible to control the possible effects of substances other than protein, when the usual high protein diet is given.

In addition to the edema a good many, though not all, of these patients complain of tenderness in the extremities, principally the legs. In spite of an absence of changes in the reflexes, sensory disturbances or significant cardiac abnormalities, this picture is strongly suggestive of a mild form of beriberi. Dr. G. A. Wheeler, for example, in discussing a report of these cases, has stated that he prefers to consider them mild or larval forms of that disease.[4] Because of this possibility a number of these patients have been treated with vitamin B preparations. The fol-

lowing products and methods of administration have been employed:* (1) A mixture of vitamin B with maltose and dextrose ("vitavose") and brewers' yeast were given by mouth, alone and in combination. (2) A concentrated alcoholic extract of rice polishings, of which one c. c. represented 10 grams of polishings, was given by mouth. (3) A purified B^1 extract, prepared from rice polishings and free of B_2, containing 700 international units of vitamin B_1 per c. c., was given orally. (4) A purified B^1 extract, containing 50 rat units per c. c. and a similar preparation containing 250 rat units per c. c., were given intramuscularly. In no case did the effects suggest with any probability that these patients had beriberi or that a deficiency of this vitamin was the principal cause of the symptoms. Tenderness and hyperalgesia were not relieved. The edema did not disappear and fluctuations which were observed differed in no way from those observed in the absence of such specific treatment. No constant effect on the serum protein levels was observed. It is realized that these results do not constitute proof of a lack of relationship of vitamin B deficiency to the clinical picture. Nevertheless, they are believed to be strong evidence against the view that the condition observed is simply a mild form of beriberi.

In the laboratory anorexia is considered to be an important and frequent manifestation of vitamin B deficiency and a similar effect has been observed in patients.[5] Although some of our patients have complained of loss of appetite, anorexia has not been an outstanding complaint, in spite of the apparent deficiency in this vitamin complex. In none of our patients has the administration of vitamin B preparations, either of the undifferentiated complex or the various purified preparations, caused any consistent and decided increase in appetite which could be definitely attributed to the specific treatment. It is also of interest that glossitis, which has been described as occurring in avitaminosis B, has been infrequent.

In a few instances additional manifestations have suggested the presence of disorders other than a simple deficiency of protein. In addition to the swelling, pain and tenderness, certain patients have shown a diffuse erythema, with increased heat, sufficient to suggest an actual inflammation. Similar symptoms have been reported in cases of epidemic dropsy in India.[6] In some of our patients a diagnosis of acute arthritis has been made in spite of the absence of other evidences of arthritis and an involvement of areas removed from joints. In other instances these symptoms have been attributed to a cellulitis or a lymphangitis. Biopsy, however, has failed to substantiate these latter diagnoses. It is of interest that we have apparently been able to produce these symptoms and cause them to disappear experimentally by increasing and diminishing the edema through variations in the intake of water and salt. Occasionally the edema has been accompanied by subcutaneous hemorrhages in the involved areas, especially the feet and legs. This, together with the other symptoms and the nature of the diet, suggests the presence of scurvy. Yet the hemorrhages have occurred in individuals whose diet was apparently adequate in vitamin C, and have persisted and recurred in spite of the therapeutic administration of an adequate diet. On the other hand, cases apparently of mild scurvy have been seen in this same group of patients and have been successfully treated with anti-scorbutics.

The occurrence of definite pellagra among these patients and the nature of the symptoms and the diet made it appear possible that in many instances the condition represented chronic forms of that disease. However, the use of general and specific measures which have proven effective in cases of pellagra has frequently failed to produce evidence to substantiate this possibility. It is true that in many instances characteristic pellagrins fail to respond to adequate dietary treatment. Nevertheless, the failure of our patients to respond to such treatment is against the view that the symptoms they presented represent primarily a mild form of what we know as pellagra.

An anemia of one type or another is a common finding in these patients. The varieties include normocytic, microcytic and macrocytic anemias, with or without an achlorhydria. Such anemias are known to occur in individuals whose diet is deficient in one or more of the substances necessary for maintaining the blood at proper levels and in some instances the anemias in these patients have responded to adequate diets or to the use of specific substances, such as iron, vitamin B preparations or the material contained in liver extracts. A considerable number, however, have been very stubborn to treatment, and some have entirely failed to respond to an adequate general diet or to treatment with one or all of

*"Vitavose" was furnished through the kindness of E.R. Squibb & Sons, New York. Preparation (3) was obtained through the courtesy of Mead Johnson & Co., Evansville, Indiana; preparations (2) and (4) were furnished through the courtesy of R. R. Williams, of the Bell Telephone Laboratories, New York.

the usual specific substances, iron, vitamin B, liver, or liver extract.

It has perhaps been somewhat unfair to select mainly the vitamin B complex to illustrate the discrepancies which may exist between experimental study and clinical application, since the confusion in regard to it is perhaps as great in the laboratory as in the clinic. Yet it illustrates well the difficulties which arise. The reasons for these difficulties and failures are many. First of all is the fact that dietary deficiencies of patients are rarely, perhaps never, single; nearly always multiple deficiencies exist. Personally, I am almost ready to believe that in the clinic none of the syndromes so definitely labeled scurvy, beriberi, pellagra, and perhaps others, are due solely to a single deficiency; rather that they always represent a combination of deficiencies.

In this connection it is interesting to review some of the symptoms and pathologic changes which characterize certain of the common and better known deficiency diseases. Edema is characteristic of one form of beriberi, yet it occurs in pellagra, in scurvy and may occur under conditions of low protein intake apparently uncomplicated by the presence of other deficiencies. Subcutaneous hemorrhages are typical of at least well developed scurvy, yet they are found in pellagra, in beriberi and in our patients with nutritional edema. Nervous system lesions are considered to be the *sine qua non* of beriberi yet they have been reported in scurvy,[7] in vitamin A deficiency[8] and are frequent in pellagra. The pain and tenderness which we have found in some cases of nutritional edema occur also in scurvy and are a prominent feature of beriberi. Gastro-intestinal disturbances are common to both pellagra and beriberi, while an anemia occurs in nearly all the deficiency states. Inanition alone has been shown, at least experimentally,[9] to cause many of the pathological changes associated with a lack of vitamin B and probably complicates the picture in other deficiency states, a fact which is not sufficiently appreciated. Under these circumstances the failure of treatment with any specific substance cannot be depended upon as a diagnostic criterion and the use of more than one specific factor leaves one in doubt as to the actual cause of the symptoms. Furthermore, we are largely ignorant of the interrelationship between the various accessory food factors and the effects of their deficiencies on the requirements, absorption and utilization of one another. It is entirely possible, for example, that under conditions of vitamin B deficiency amounts of vitamin C which would otherwise be adequate become insufficient. In a similar way deficiencies in intrinsic substances, similar to that concerned with the production of pernicious anemia, may influence the requirements and utilization of accessory food factors.

Another difficulty in the clinic, and one which is closely related to the question of multiple deficiencies, is our inability in clinical studies to control the diet. This occurs both with respect to our knowledge of the dietary defects which precede the onset of symptoms and the use of diet as a therapeutic test of diagnosis. Variations in the quantity as well as the kind of food, the effects of cooking, individual variations in requirements which result from work, exercise, metabolic disturbances, and the like, are difficult to determine accurately. Because of the complexity of diets it is difficult to supply food which is acceptable or accessible to the patient and will provide single specific substances to the exclusion of others and at the same time maintain nutrition so as to avoid the effects of inanition. Finally, structural and functional changes which may have resulted from the deficiencies, or preceded them, may affect absorption and utilization, thus interfering with the response to specific treatment and causing confusion.

If these difficulties exist in the case of the more manifest disorders they are much greater in the case of the latent or subclinical disease. There the vagueness of the symptoms, and the atypical signs, their similarity in one and another of the deficiency states and in diseases not related to diet, their spontaneous appearance and disappearance as the individual wavers between good and ill health, depending on a multitude of factors, all make the precise recognition and adequate treatment of these disorders most difficult.

If this discussion has seemed somewhat pessimistic it was not intended to be so. Instead it was the purpose to point out some of the difficulties involved in the clinical application of knowledge obtained from the laboratory and indicate some of the advances necessary to a better understanding of deficiency disease in humans. Among the latter is the need of developing pathognomonic signs of the deficiency of single food factors, such as the rate of regeneration of the visual purple as a test of vitamin A deficiency.[10] A second, and a corollary to the first, is the development of purified preparations which may be used with the assurance that the effects obtained represent the action of a single

factor only. The preparation of crystalline vitamin C (ascorbic acid) is an example of a recent notable advance in this direction.[11] A third is the need for preparations which can be administered by routes which will avoid the difficulties and uncertainties of absorption. Finally, there is required a critical and scientific attitude in the evaluation and interpretation of the methods and results of clinical studies in this field.

SUMMARY

In summary, then, attention has been called to the difficulties and uncertainties which arise when one attempts to transfer knowledge gained from animal experimentation to the diagnosis and treatment of dietary disease in patients. Examples of these difficulties have been presented from the study of a group of patients whose diets are known to be inadequate in various respects. These patients present symptoms which are believed to be due to dietary deficiency. The symptoms often suggest the presence of at least mild forms of some of the better known deficiency diseases. In many instances, however, it has been impossible to establish definitely the nature of the deficiency. Some of the causes of this difficulty have been discussed, in particular the importance of multiple deficiencies, and some of the developments which are necessary to further progress in this field have been mentioned.

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DISCUSSION (*Abstract*)

Papers of Dr. Musser, Dr. Forbes and Dr. Youmans.

Dr. W. R. Houston, Augusta, Ga.—The remarkable progress made in the last two decades in our knowledge of vitamins is a striking illustration of the effectiveness of the weapons that science now possesses. As A. N. Whitehead points out, "The greatest discovery of modern science is the discovery of a method by which scientific discoveries may be made." We are no longer dependent on the inspiration and the intuition of the occasional genius. When the subject of these accessory foodstuffs, the vitamins, was attacked it was known at once that a method for quantitative assay of these values must be devised. In laboratories everywhere in the world workers set themselves to the task of devising technic for studying these substances quantitatively, looking ultimately toward securing them in such pure chemical form that their properties could be analyzed free of extraneous admixture. The studies made, at first hesitant and stumbling, have gained in speed and momentum until we feel that now we are on the verge of being able to answer many of the questions about vitamins that naturally suggest themselves.

Some of the most important of these questions, however, are not answered yet. Our knowledge at present is limited largely to an estimate of the irreducible minimum of vitamin supply that will safeguard against deficiency symptoms. Besides this knowledge we should like to know what is the optimal amount of each vitamin for an ideal diet. We should like to know whether an administration in unusually large dosages of this or that vitamin can be advantageously employed against situations in which pathological physiology has developed. We should like to know whether advantageous stimulation of bodily function can be obtained through the obscure forces that are concealed in the vitamins. We should like to understand in detail what is the chemical mechanism by which the vitamins produce their effect on metabolism and function. The answer to many of these questions seems to lie close at hand, since some of the vitamins have already been isolated in pure chemical form, their structural formulas worked out, and we can but believe that in a short time we shall have an accurate chemical knowledge of all of them so that it will be possible for a person living on an avitaminous diet to take all the vitamins required for his daily needs in a single small potion.

It is evident that the knowledge already acquired concerning vitamins has already influenced our dietetic habits. Whole industries have been affected by these scientific researches. Feeding plans for infants, in particular, have been greatly changed. It can hardly be doubted that our newer knowledge of these accessory foodstuffs has led to the preservation of many lives and the lifting to a higher level of still more.

While all this is true, it should not be forgotten that the vitamin question is not the most important of the aspects of diet. We must not permit the charm of novelty to give to the vitamin problem unwarrantable weight in our consideration of dietary problems. The first question about a diet is whether it contains optimal nutritional value, whether it has calories enough. James J. Walsh, after a long life spent in successful practice and treatment, wrote not long since that in looking back over his work he considered that the most striking

and successful cures that he had ever made resulted from giving undernourished people enough to eat. Some students of diet, impressed by the difficulty experienced in endeavoring to construct a diet that excludes a vitamin, have rather insisted that if one has enough to eat of foods that are palatable he can scarcely avoid getting all the vitamins that he needs.

It should not be inferred, however, that instinct will lead creatures to choose foods containing an appropriate proportion of vitamins. The liking for foods that are rich in suitable vitamins is not instinctive, but a conditioned reflex, as has been shown in the laboratory. If an essential vitamin is placed in a certain food and the animal habituated to taking this food and then later the vitamin is withdrawn from this article of diet and placed in another to which the animal is unaccustomed, he will continue to eat the food without the vitamin until he becomes sick and wasted; he must become reconditioned to a liking for the food that contains the life preserving vitamin.

It is excellent that we should be given an opportunity in a symposium like this to keep abreast with the rapid advances being made in this interesting field, a field so large that it can be reviewed only with great labor, as Dr. Musser has pointed out. We have enjoyed here an opportunity to hear a scholarly appraisal of all that has been done up to this moment.

THE COLON*

A CONSIDERATION OF ITS IMPORTANT DISEASES AND DISORDERED FUNCTIONS

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Diseases of the colon are relatively few in number. The actual separation into independent groups, organic disease on the one side and functional disorders on the other, is a difficult task.

There are many patients with border line conditions which cannot be clearly classified, because the clinical symptoms are out of all proportion to the physical and laboratory findings. If, however, the border line conditions are studied over a long period, the number of cases falling into this group will be increasingly diminished.

The correct approach in such a study should be the differentiation between actual disease and functional disorders.

Diseases of the colon may be classified into four main groups:

(1) New growths
(2) Parasitic diseases
(3) Infections
(4) Anomalies

Considering the relatively few diseases comprising this group, the diagnostic approach is not very difficult. The symptoms are often pathognomonic. This is particularly true in parasitic diseases and new growths producing obstruction of the bowel.

New Growths.—The majority of tumors arising in the colon are malignant in nature. Ewing[1] classifies cancer of the colon and rectum into six pathological divisions. Clinically, two main types are observed: the large cauliflower growths in the proximal bowel and the small, napkin ring stenosing tumor of the distal bowel.

The symptoms may be divided into two groups: those due to obstruction and those due to degeneration and absorption.

Tumors[2] in the right half of the colon present large, elevated, ulcerating surfaces, bleeding easily and lending themselves readily to infection, but they rarely produce obstruction. Tumors in the left half usually invade the lateral wall of the bowel and encroach upon the lumen, frequently producing obstruction.

The symptoms vary according to the degree of obstruction. Unusual rumbling of gas or a feeling of fullness in the abdomen is often the first symptom. As the tumor increases in size, it delays the fecal stream, but for a time, compensation is effected by the dilatation of the bowel above the lesion. As the constriction increases, peristalsis becomes more vigorous and colicky pain results. The pains are accompanied by the rumblings of gas and are increased by purgation. Further progress of the obstruction produces visible peristalsis during the attack of pain. This is characteristic of obstruction.

When the tumor begins to degenerate, slight bleeding occurs. As the ulceration progresses, larger hemorrhages will present sufficient blood to be observed macroscopically.

The septic material from the ulcer increases functional activity and diarrhea occurs. Brown[3] reported the occurrence of diarrhea in 36 per cent of his cases. In our series[2] it was observed in 46 per cent.

Loss of weight and anemia are mild in the early stage but are severe as the ulceration progresses. The growths in the proximal loop produce anemia and cachexia earlier than those in the distal segment. Carcinoma of the cecum is bathed in the most highly infectious fluid found

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in the body. This fluid, teeming with bacteria, is thought to be the chief factor in the production of anemia.

The diagnosis of neoplasms of the colon is made by the symptoms, the roentgen evidence of deformity after the opaque enema, and the sigmoidoscopic view of the lower lesions. The proctoscope permits direct visualization of the growths in the rectum and rectosigmoid. Sections for biopsy can be easily removed from lesions below the sigmoid by a specimen taken through the proctoscope.

Parasitic Diseases.—Infestation of the colon by intestinal parasites is such a common occurrence and has been given such wide publicity, particularly since the Chicago epidemic, that stool examinations have become almost a routine procedure. The chief organisms producing disease are the *Entameba histolytica* and the dysentery bacilli.

The symptoms vary from a mild intestinal upset to the violent constitutional upheavel seen in acute colitis.

The diarrhea of amebiasis is characterized by frequent evacuations of bloody mucus, with small particles of feces. The stools, on microscopic study, reveal numerous blood cells but very little pus. The amebae contain ingested red blood cells.

Bacillary dysentery involves the entire colon. There are no isolated ulcers as are seen in amebiasis. The stools are thick, purulent and foul smelling, consisting principally of pus and endothelial cells mixed with feces. The diagnosis is made by the culture of the stools and the presence of specific agglutinins, as shown by the Widal test.

Infections.—Chronic ulcerative colitis, tuberculous enteritis and diverticulitis, constitute the infectious conditions of importance.

The salient features of ulcerative colitis are a continuous or intermittent diarrhea, abdominal pain, anemia and general debility. The mucous membrane is hyperemic, edematous, granular and bleeds easily. A profuse exudate covers the lining of the bowel which is later replaced by superficial and deep ulceration. The infection usually begins in the rectum and extends upward. The ulcers are confluent with shaggy, indefinite borders. The symptoms, the characteristic proctoscopic picture, the rigid, tubular, and sometimes string-like roentgen appearance of the bowel, suggest the diagnosis.

Tuberculosis of the colon may be a part of a general tuberculous enteritis. Examination by the opaque enema reveals localized deformity, irritability and spasm in the proximal colon with ileocecal insufficiency. Nine hours after a barium meal, there is a delayed emptying of the ileum, non-filling of the involved proximal colon and a rapid filling of the colon distal to the lesion.

Diverticulosis is commonly seen in robust, stockily built individuals. The diverticula may involve the entire colon but they are more numerous in the sigmoid flexure. When inflammation occurs in one or more of the diverticula the symptoms are fairly characteristic. There is pain in the left abdomen with a feeling of fullness, extreme soreness, gas and usually diarrhea. Examination reveals exquisite tenderness over the inflammatory area.

Perforation of a diverticulum with local pericolitis may be confused with carcinoma because of the deformity revealed by the opaque enema. The acute symptoms, the presence of other diverticula, the longer duration of symptoms, the infrequency of bleeding in diverticulitis, are the classic points in differentiation.

Anomalies.—Anomalies of the colon are a distinctive expression of organic constitutional inferiority of the digestive tract. They are of importance clinically because they are frequently encountered and they predispose their bearers to colonic malfunction. Kantor[4] found the colon redundant in 19 per cent and the cecum low in the pelvis in 18 per cent of one thousand gastrointestinal cases studied.

The symptoms associated with redundant colon are constipation, dull dragging pain, usually in the loins, dyspepsia and gas distress. If the cecum is low and fixed in the pelvis, the ileocecal valve may be incompetent and the symptoms are those of so-called auto-intoxication, namely: nausea, vomiting, headache and generalized aching.

These conditions are of interest because of their frequency and because of the many medical and surgical measures which have been instituted to correct the deformities. Few of the methods have been successful. Fortunately, a large per cent of these patients get along fairly well when dietary and eliminative measures are properly enforced.

Functional Disorders.—The diagnosis in these cases is often most difficult and requires the

maximum of patience and industry. It is necessary first of all to eliminate organic gastrointestinal disease by every means available, to consider all the extradigestive tract lesions that secondarily produce gastric symptoms, such as disease of the cardiovascular, renal, respiratory and central nervous systems; then to analyze the patient's mental and physical make-up, his environmental factors, his social and financial status. Only on such a broad basis is it ever justifiable to make a functional diagnosis.

When we are certain we are not dealing with organic disease, it becomes necessary to find a satisfactory cause for the disturbance in function.

It seems that a working classification according to etiology would be more expressive of the underlying factors which result in disturbed function. Such a classification could be expressed in the following groups:

(1) Neurogenic
(2) Allergic
(3) Dietetic
(4) Reflex

Under the neurogenic heading, Hartsock[5] has clearly shown four groups of patients with a nervous instability as a background and whose symptoms are referred to the digestive system.

Group I.—The first group is of those who are constitutionally inferior and who are entirely inadequate to cope with the ordinary problems of life.

Group II.—In the second group are placed the mentally active and ambitious, who have a poor physical constitution by hereditary or environmental influences, such as some nutritional problem, childhood tuberculosis, or other chronic illness during the developmental period. They get along fairly well in life for a while, but do not realize the limitations of physical strength and their ambitions lead them into a breakdown with resulting intestinal symptoms.

Group III.—The patients in the third group have a normal constitution, but through sickness, repeated pregnancies, overwork, domestic and financial worries have become completely exhausted. These patients lose their appetites, suffer with indigestion, constipation, abdominal distress and often develop anemia.

Group IV.—The fourth group includes those with sound constitutions, who are inclined to be erratic and are emotionally unstable. They are irritable, emotional and suffer with cold hands and feet, indigestion and alternate constipation and diarrhea.

Patients falling into either of these groups may seek medical advice because of intestinal symptoms. It is only by conscientious effort and sympathetic questioning that the real cause may be found in the background of a disturbed nervous constitution.

Unfortunately, there is no specific treatment for these patients and they become "colon minded". They usually go from doctor to doctor seeking a remedy which will relieve them of their abdominal soreness, constipation, diarrhea or mucus. They take all the remedies for amebiasis and for colitis. They get no better, but strange to say, they rarely grow worse.

It is my custom to ascertain the background of these patients, seeking the spring which started the flood of nervous impulses, resulting in the disturbed function of the intestinal tract. If this spring can be found and diverted back into its normal channel, restoration of function may sometimes be accomplished.

Allergic.—Diarrhea and constipation caused by food allergy are due to a localized congestion of the intestinal mucosa, similiar to the bronchial spasm in bronchial asthma. It is not surprising that smooth muscle spasm, mucous membrane irritation and edema due to food allergy occur in the intestinal tract, especially in the colon, where the food residue frequently remains in contact with the tissues for two or three days.

Dorst and Morris,[6] in a study of the bacterial flora of the colon, found that many patients with irritable colon reacted positively to the ordinary intestinal bacteria. A large number of these patients were benefited by desensitization with autogenous vaccines.

Careful skin testing with foods and possibly bacteria may reveal the cause of some of these obscure intestinal disturbances.

I have a number of patients who were relieved by eliminating from the diet foods which gave positive reaction on skin testing.

Dietetic.—Functional disorders of the bowel may be due to an unbalanced diet. Excessive fats and sweets frequently produce diarrhea with alternate constipation. Nature has equipped the digestive organs with enzymes to digest certain amounts of carbohydrates, protein and fats. If the food intake does not conform to the normal output of one or more of the enzymes, the result is improper digestion and malfunction chiefly in the form of colonic stasis.

Reflex.—For a number of years I have been impressed with the large number of patients with gallbladder disease who complain of intermittent diarrhea and those with chronic appendicitis who have obstinate constipation.

The diarrhea of gallbladder disease may be due to the irritating effect of an excessive flow of bile or to a reflex stimulant originating in the upper intestinal tract, increasing peristalsis and resulting in hypermotility of the lower bowel. Whatever the explanation, the diarrhea usually disappears after the infected gallbladder has been removed. The outstanding symptoms in chronic appendicitis are, constipation, soreness in the right abdomen, and indigestion. A study of the histories of a large number of these patients reveals the striking fact that the onset of constipation frequently followed the first attack of colic. The roentgenolic study will often reveal a marked stasis and spasticity of the colon. After the appendix is removed many of these patients return to a regular bowel habit and the colon assumes its normal function.

There are other functional disturbances of the colon which are associated with and form a part of chronic constitutional disease. Metabolic diseases and blood dyscrasias not infrequently have as part of their symptoms diarrhea, constipation or flatulence. These conditions, however, are primary and the intestinal symptoms will disappear when the cause has been removed.

Patients with organic disease of the colon are easily satisfied when we can show them the cause of their trouble, by displaying x-ray films and laboratory reports. It is true the treatment may not offer complete relief, but they know the cause has been found and a definite treatment can be instituted. This fact, coupled with the confidence of the patient and the skill of the physician, offers hope of recovery.

Patients with functional intestinal disorders, however, present an entirely different problem. The digestive symptoms of the constitutionally inferior, the visceroptotic, the emotional, the unstable and the mentally harassed patient, cannot be explained by the x-ray, the laboratory or the physical examination. We must go beyond the physical, to the mental, the moral, the domestic, the social and even to the developmental background to find the facts which disturb the normal functions.

The science of medicine and surgery is at its best when dealing with organic disease. In functional disorders, we must call into service our training and experience in the art of medicine to relieve the anxieties, dispel the fears of harassed patients. We should use fewer drugs and restricted diets and confine our efforts chiefly to restoring function by the same channels through which it became distorted.

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CONSTIPATION AND BOWEL TRAINING*

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This subject is one with which every physician is daily confronted and on which the advice given or the treatment prescribed is very varied, so that patients who may have occasion to consult more than one physician will often find themselves in a true dilemma and frequently end in a state of confusion rather than enlightenment as to the true state of affairs.

Health should be the first consideration and any condition thoroughly compatible with good health in the particular individual can hardly be considered abnormal. It is quite true that babies on an adequate diet who have no more than three stools a week are for the most part among the well nourished, vigorous, contented and happy group in which we feel a true pride.

The real question, then, is: "What is a normal diet, and what is a normal bowel function in any individual case?" The same diet in different individuals of the same age and size, as for instance twins, will often produce a very marked difference in physical progress and in bowel function. For this reason it is difficult, indeed, to set down rules of diet which will not have to be altered in many instances to meet the individual requirements of particular patients. On the other hand, the number of stools per day or per week in children on identical diets will vary and we again are unable to lay down any simple criteria for the normal as a guide in this determination. As before stated, good health of the individual should be the first consideration.

*Read in Clinical Session, Texas Pediatric Society, meeting conjointly with Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

Two factors primarily are concerned in bowel activity: the digestive organs as a system and the food content of this system. To these should be added habit and certain miscellaneous factors as physical activity, fatigue and posture. There are conditions influencing the passage of the intestinal content in which so-called constipation is a prominent symptom, such as congenital anomalies of the digestive system, cretinism, Hirschsprung's disease, Mongolian idiocy, pyloric stenosis, visceroptosis and spastic colon, which, however, include only a small number of the cases presenting and which do not constitute the big problem for solution. Time will not permit a discussion of all of these conditions.

In this discussion our concern is the case in which none of the above major conditions are found, and in which the only complaint is constipation or inability to regulate the bowel. In other respects the infant or child, as the case may be, is reasonably within normal limits.

Since constipation is the complaint, what is meant by this term? In some cases it means that the stools are large and firm, although there may be two or more passages a day. In others it means that there is no more than one small passage a day and the mother has been told that in infants or small children at least two evacuations daily must be had. In some cases it means that the mother has used some artificial means each day to evacuate the bowel because, due to the baby's fretfulness, she was afraid to wait to see whether a spontaneous movement would occur. In another case it is because the child does not go to stool each day, although the passage when occurring is of normal consistency and size. Many variations from these situations are reported as constipation, as you all know, most of them being adjudged by arbitrary standards in the mind of the mother or grandmother. These people throw up their hands in horror when they learn of a breast-fed infant who has gone many days without an evacuation of the bowel and shows no signs of injury.

I am unable to give a satisfactory definition of constipation even to myself, but to me it should represent a definitely abnormal state in the child, with unpleasant symptoms to the child and not the mother, and I am confident that whatever the definition may be, many of the cases grouped under this category are in reality not abnormal in any way. Few will disagree with the statement that the frequency, character and size of the stools will vary with the quality and quantity of food ingested and retained over a given period of time. If, then, we start with the fasting individual with no stools at all and go up by steps to the gourmandizer, we must of necessity have a wide variation in the number of stools in any given time. On the other hand, we can take the same individual who has a perfectly regular and satisfactory bowel function, let us say of one stool a day, and by variation of his diet reduce the number of stools to three or even two a week without particularly unpleasant symptoms to that individual. Is he then to be considered to be suffering from constipation simply because of this change? If his diet of the pre-experimental days is resumed, the bowel function is again the same. Admittedly there is nothing wrong with the bowel in this case; therefore how can constipation actually exist? It is a matter of regulation of the diet, not of the bowel, as is the usual statement by the mother.

In the breast-fed infant the stool is always soft whether there be five stools in one day or one stool in five days. There is never a dry, hard, formed stool and except in cases with a very tight anal sphincter, defecation is always easily accomplished. Therefore, can we say that a true state of constipation ever exists in the case of a breast-fed infant? If the supply of breast milk is adequate and the infant ingests sufficient of this, the bowel movement will, as a rule, be satisfactory to even the most critical mother. Where the mother comes in with a complaint of constipation in a breast-fed baby I have usually found one of two situations responsible for the failure of the bowels to move one or more times each day. The first of these is that the milk supply has not increased normally with the growth of the child and is therefore inadequate to meet the requirements of the child at the particular age and so little residue remains that proper stimulation does not occur until after the accumulation of several days. The other condition is that where the infant is of the extremely healthy, fast-growing type with an unusual digestive capacity resulting in the digestion, absorption and assimilation of such a high percentage of the food ingested that again too little residue is left to produce stimulation of the reflex daily. In these cases additional food for the first child and education of the mother in the second case is all that is required to take care of the situation.

The infant on artificial food, on the other hand, does and should have a formed stool, but to the mother accustomed to the type of stool

seen in breast feeding, this stool immediately represents a state of constipation and such a complaint is registered. Now, there is no change in the bowel function of such an infant overnight or over the week, and it is foolish beyond degree to let this mother begin at once with laxatives, suppositories, enemata or what not to regulate the child's bowel. The whole factor responsible for the change is the intestinal content, not the intestine itself. Therefore, the mother should receive a rational explanation for the change, instruction as to the alterations in diet, education in proper habit formation, and above all reassurance as to the true state of affairs. It is not necessary to attempt changes so great as to secure a soft stool, nor is it even advisable to do this, especially if the use of lubricants or laxatives is resorted to.

By beginning early with artificial irritants a mother can often so interfere with the development of the normal reflex as to bring about a permanent change, even to the point of doing irreparable damage. The child gradually gets to the point where he expects and relies on this practice for help and either becomes insensitive to normal stimulation or unable to respond without help. In the older child who for a number of years had a fairly normal bowel activity and at an advanced age shows up with a complaint of constipation the almost universal cause is found in the diet with other factors contributing in lesser degree. A change in eating habits, being permitted late hours at night, school rush, poor posture resulting from these and many other new conditions may have an influence.

First to consider is the child who for reasons of attracting attention, causing anxiety, or gaining some point of personal privilege refuses to eat or begins to vomit his food. No longer is a child's obedience maintained by being "put to bed without any bread." On the other hand, the child too often knows that he holds the whip hand over his parents and by his threats not to eat or to vomit if he does eat, completely reverses the program and the parents are the ones who accede to the child's demands. This is all too true in the one-child family and constitutes, I believe, the greatest single problem on which I am consulted. Many times this child, when placed in an entirely different environment without directing his attention to the change, will behave quite differently and need no further help to the solution of his difficulties.

As in other conditions, treatment should be discussed under the heads of:

(1) Prevention and

(2) Active treatment for the individual case.

Early measures directed at prevention of the development of a state of improper bowel function and the avoidance of the use of intestinal or rectal irritants in either treatment or training will do much to cut down, in later years, the number of patients with a complaint of constipation. The normal stimulant to intestinal activity should come through the diet as opposed to intestinal irritants in the form of medicines, soap sticks, suppositories, and so on, and too much cannot be said against the use of these latter in the program of the otherwise normal infant or child.

In the discussion of the methods to be used in handling these cases, habit training is among the first in importance and applies to all ages in the same way.

First, habits of regular nature in eating and sleeping should begin with the small infant and should be just as carefully controlled in the older child. Next, some systematic effort at bowel training should be begun early and, even in those cases in which imperfect results are at first encountered, should be continued until the child learns the reason for the training effort and responds. Then later still, good habits in mastication of food, in eating time, in play and outdoor exercise are all valuable as aids. Improper food habits in the parents set many bad examples for the child and the frequent statement that he is too young for this or that usually engenders sullenness and resentment on his part and results disastrously.

The second consideration next to habit training would be an ample diet. Many variations in diet may produce equally good results, provided such diet is sufficient in quantity, but there are certain factors common to all adequate diets which must be understood by the mother so as to guide her in her daily routine. The earliest instruction to the mother is to avoid those foods which are not essential in the diet and which might by reason of preferable flavor cause the child to turn against the "staple" foods which he has enjoyed well enough up to this time. Mothers seem to have an inordinate desire to introduce into the diet ice cream, candies, fountain drinks and highly seasoned foods before the child has sufficiently well established food habits to assure his continuing in a normal way with his food.

Our experience is that the difficulty is not in getting the mother to do enough for the child, but rather to keep her within bounds and not doing too much.

Beginning with the new born infant, if breast-fed and making a normal gain and contented, there is no need to concern oneself with the amount of food being taken or with the number of stools being passed. If the stools are infrequent and the gain slow, then a satisfactory complementary feeding is given. However, bowel training is begun early in all cases and it is not at all unusual with us to have a mother report when the infant is two or three months of age that he is using a pot and not soiling the napkin. A little later, when the child can sit alone, a nursery chair is introduced and continued. Frequently a break may come in the program due to an illness and unless the habit is well established a resumption of the training program will be necessary to get the child back to regular habits, and it is in the failure on the part of the mother to realize this need that we find the basis for many of the cases referred to above which have developed so-called constipation after a period of years of satisfactory bowel function.

The young infant on the bottle having a formed stool will of course experience more difficulty in defecation than a breast baby, and since by nature the intestinal tract is prepared only for a soft stool and the abdominal muscles are as yet relatively weak, training in the bottle-fed is even more important with especial reference to the development of the abdominal muscles. Where new or more strenuous muscle function is required of an individual, training and development are always necessary and the young infant is no exception. If medicines are given to produce soft or liquid stools development and training are interfered with and often delayed even to the extent that harmful results follow.

The technic of bowel training is as variable as any procedure that I know of. Many mothers profit from accidental discoveries, as for instance the use of a thermometer in the rectum, while others by trial and error methods learn a way in which satisfactorily to stimulate the reflex. In cases where no effort has been made or no results have been obtained by the method in use, it is our custom to suggest to the mother these steps:

(1) Pressure against the anus with the tip of the index finger. If no results with this, follow by

(2) Insertion of the tip of an ulcer syringe lubricated with vaseline.

(3) In either case when the child shows a disposition to strain hard, place a vessel in contact with the buttocks so that the stool is passed into this. In this way a conditioned reflex is usually established fairly promptly and in a short time only the contact of the vessel against the buttocks is needed to bring about results.

If a daily bowel movement is to be expected the diet must be ample, and not only this, but the child must ingest sufficient food to meet all his requirements and leave a normal amount of residue, and this must be stimulating in character, not, as we term it, "flat." A high roughage content, as has in the past been so universally recommended, is not necessary, and even in some cases may work to a disadvantage. However, it is important to keep up a proper proportion of such foods and to avoid excesses of those foods which are capable of too complete digestion and absorption. Older children with a high intake of milk will often show immediate improvement if milk is cut down or omitted entirely from the diet. I am led to conclude that in many of these cases milk itself is not constipating as is usually claimed, but the intake of other foods is so low when much milk is ingested that a proper balance and proper residue is not maintained.

In the diet of all such cases coming to us we stress the following points:

(1) *Fluid Intake.*—We insist on extra fluid with a full glass of water on arising each morning.

(2) *Food Intake.*—The American public neglects breakfast, and this is an extremely important cog in the machinery, as a full meal following a fast of several hours is an important factor in stimulation of the entire intestinal tract.

(3) *Vitamin Intake.*—I have been impressed with certain cases in which the vitamin content of the diet, especially vitamin B has been increased and very little alteration made in any other respect with the result that a marked difference in the character and the number of stools follows. There seems to be much to suggest that the trouble in the child on a low diet may be due to avitaminosis, as frequently in these cases the reduction in diet is more at the expense of the high vitamin foods.

(4) *Special Content of the Diet.—Beta* lactose has been an aid in many of our bottle-fed infants and is to be recommended in all cases. Black ripe bananas have been among the best aids at our disposal. Practically an unbroken series of successes can be shown where it has been possible to get the proper cooperation of the mother. By "black ripe bananas" is meant exactly what the term implies. Not a brown or speckled peel and not a banana that is sour or

blackened from bruising. It is often necessary for the mother to buy the fruit ahead for many days in order to avoid interruptions in the program. From one to three bananas a day may be given as desired, preferably at the end of the meal. This gives something that most children like and has even been used as a means of encouraging the child to take other things that will be beneficial.

The part of the mother in handling these cases is of major importance and the mother who comes to the office expecting a treatment or diet which can after a week's use "regulate the bowel" to such a degree that no further special attention is required and so that any diet will in the future serve the purpose is of course unreasonable. But that is what many expect of us, and if the mother cannot be made to understand that each 24 hours is a separate period and calls for its own program, success cannot come regardless of the suggestions offered. No more can enough food be taken in one day or one week to promote a continued proper function of the bowel than can enough fluid be taken today to insure proper bladder activity next week. However, in time the habit element becomes more and more the predominant thing and when sufficiently well impressed on the child a more varied routine may be permitted without disturbance of function.

The attempt has been made here to enter a plea to the profession on behalf of those children who are essentially normal, but who will in many instances come to a truly abnormal condition because of the use of abnormal intestinal irritation instead of normal intestinal stimulation.

THE IMPORTANCE OF THE PRIVATE PHYSICIAN IN A PUBLIC HEALTH PROGRAM*

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The development of preventive medicine and its close association with curative medicine may be traced back many hundred years. In this development it is interesting to note, and we should be justly proud of, the prominent part that physicians have played in the evolution of our present day practice of public health.

*Read in Section on Public Health, Southern Medical Association, Twenty-Eighth Annual Meeting, San Antonio, Texas, November 13-16, 1934.

Almost without exception, the men who have done most in developing the health departments to their present state of efficiency have been physicians. Bacteriologists, physicists, chemists, nurses, and research workers who were not physicians, however, have discovered many facts in preventive medicine, but it was the physician acting as health officer who applied this knowledge and developed the system of wholesale preventive medicine which is the main objective of health departments. No significant collective action in preventive medicine by organized medical societies was in evidence except in the larger cities until about 1915. The development of preventive medicine in health departments since 1915 has been rapid, through the vigorous efforts of organized health workers. Even more enthusiastically, unofficial agencies, by educational propaganda, have insisted on prevention and the development of facilities for prevention rather than cure.

These unofficial agencies were able to secure large sums of money for such preventive work, educational and otherwise, and the official health officers secured for their departments large appropriations to prevent diphtheria, typhoid fever, tuberculosis and other diseases. As a result, an artificial gulf developed between the physician who was a health officer and practiced preventive medicine and the physician in private practice who practiced curative medicine. This gulf should never have been created and, fortunately, is now disappearing.

The only excuse for invasion of the physician's territory was that the physicians, individually and collectively, would not do those things that were urgently necessary. If we were to accomplish anything in preventive medicine, no health officer could sit idly by while children died; incipient tuberculosis became advanced tuberculosis, and readily controllable diseases ran rampant; when aggressive action, even if wrong in principle as an invasion of the private physician's field, could prevent this unnecessary loss of life.

It was not until this country entered into the World War and one-third of the men who applied for, or were drafted into, military service were found to be physically unfit, that the eyes of the nation were opened to the necessity of producing a more nearly perfect race. President Hoover realized this national need and called the White House Conference for the Health and Protection of Children which met in Washing-

ton for several sessions during 1930-1931 and which was participated in by over a thousand trained workers from every section of the United States and the insular possessions. One of the important factors resulting from the mass of information obtained was that medical students and nurses were not getting sufficient pediatric training and physicians were not practicing preventive medicine among their clientele.

Until our medical students and nurses go out for service with a better preparation for this specific kind of work, they will suffer an unjust handicap in their efforts toward maintaining health and preventing disease. The medical faculties, and especially the clinical teachers of every medical school, are rapidly coming to realize that the medicine of the future is to be in large part preventive medicine, and it is their duty to prepare their students for service along these lines.

The principles of preventive medicine are sound and the visualized result is the development of sounder physical and mental manhood and womanhood. Upon this depends our very national existence, and one may honestly appeal to the patriotism as well as to the common sense of the American medical profession to rush forward this very necessary piece of work, with resulting benefit to their country and to themselves.

Many physicians have always been earnest and helpful in the application of preventive measures and have applied them for generations but these measures can no longer be carried out by the physicians alone. In order to reach all the members of the community, mass measures must be applied by the health department.

The medical practitioner has a real place in the public health field. As a matter of fact, as time goes on preventive medicine is going to consume an even greater proportion of his time. This does not mean his practice of those ordinary things that are expected of every conscientious doctor, such as giving prophylactic biologic preparations, making efforts to protect the community against the infection of his patient, and helping in various clinics for the indigent. These he does now to a greater or less degree. But preventive medicine has taken on a broader meaning in recent years. There has come a realization that humanity can be made happier by the early detection and correction of minor deviations from normal than by curative measures applied after these conditions have produced gross pathology. The family doctor, better than anyone else, can, if he will, make the next generation more fit than the present. He cannot accomplish this if he neglects to immunize his own children, his clients and their children, and those who have entrusted themselves into his care and keeping against preventable diseases, or fails to keep these children free of physical defects which are now found in such numbers in the practice of many physicians that one wonders if any preventive efforts at all have been practiced.

Opportunity is in the making for the private practitioner to take his own peculiar place in the public health movement. Technical preparation there must be, exactly as one would fit himself for any other special procedure. A new approach, a new viewpoint must be acquired by some. The general practitioner, if he cares to apply himself, is able to do this work, and he has only impeded its progress, as well as held himself back, by a lack of appreciation of his most important place in this program. He is urgently needed and should have the assured confidence and backing of those who understand this work and wish to see it progress. He has readily been able to accommodate himself to the more difficult curative aspects of medicine, and his ability to do this work well can be measured by his relative standing and conscientious application to the principles of curative medicine. It is impossible to tell how many patients are actually being sent away from the door of the general practitioner by the doctor himself, because of the fact that he has done so little to justify the belief that he is aware of the virtue of preventive work and that he is able to give to the people the very service that

they go to the health department to obtain. When the rank and file of general practitioners manifest a sound interest in this important field of medicine, and only when they do, will this work make the progress that the leaders of the medical profession wish it to make, and it behooves the outstanding figures of the American medical world to preach this gospel through the medium of national, state, county and local medical societies.

An idea seems to be entertained by some physicians that the family physician is the only man who is competent or has the right to discuss matters of health. It is granted that the family physician is better qualified to assume leadership in many phases of this movement, but it is the height of folly for him to decline to cooperate cordially and continuously with the duly elected health officers, public health nurses, inspectors and other agencies that are working in this field, not only in the legal requirements as to reporting births, deaths, cases of communicable disease, and the like, but in the matter of arousing the citizens of the community to the importance and great responsibility of preventive measures. However, it is the right of the private phyisician to insist that the activities of each of these groups of workers be confined to programs outlined and approved by the state board of health, the membership of which in most states is composed of private practitioners of medicine and surgery who have at heart the best interests of the profession and of the people, and who, themselves, are selected by the state medical association to be responsible for the proper conduction of health programs.

The relationship of the health officer to the physician should be one of friendliness, mutual helpfulness and genuine whole-hearted cooperation in everything which pertains to the health and prolongation of the lives of the citizens, because it is only in this way that each of them can best succeed in the performance of the duty that devolves upon him.

The success which any health officer attains in the performance of his duty will be very largely dependent upon the support and cooperation which he receives from the physicians in the community over which he has jurisdiction. This is an axiom which all health officers must recognize and observe.

One of the chief criticisms to be made of preventive work, as done by health officers, surgeons, physicians, and dentists is that they do not do nearly enough of it. The more the work is developed, the more of it there is to do. In communities where there are no preventive programs, where the physicians show little interest in prophylaxis, there is little or no demand for such service; but where preventive programs are common and the physicians interested, the demand increases steadily.

The public is increasingly coming to accept and appreciate disease prevention services, but it is not yet ready to pay for preservation of health as it pays for health restoration. Further education and demonstration of the value of preventive measures by health departments and private organizations are necessary. Medical societies themselves must take a more active part in this work. Education in health preservation has been intensively carried on by private organizations and health departments for less than two decades, and in the schools an even shorter time.

The organized profession has been seeking the education of the public for some years by supplying authentic information on health topics to newspapers, magazines, journals and other publications; by the preparation of pamphlets; through the medium of lectures and radio talks and by cooperation with other national organizations interested in a movement for better health. The American Medical Association has sought to educate the public to a better understanding of the purposes and ideals of the medical profession through the establishment of *Hygeia,* a journal of individual and community health, published monthly. The giving of health advice has not always been whole-heartedly supported by the medical profession. Advice given by others has often been conflicting and some

has been fraught with error, which is an urgent reason why physicians should assume the lead in public health education. The keynote of health education by lay groups is, and should be, "Consult your family physician."

Much unreliable commercialized health advice has been given over the radio in recent years. This advice has, fortunately, in many cases been so ridiculous as to make its serious acceptance by the public unlikely. The medical profession should seek some form of radio censorship, such as is now in effect in many published advertisements, which would gradually drive out misleading statements now often heard.

In the field of preventive medicine, the health officer prepares the way by education and demonstration for the physician who should make ready and avail himself of the opportunity for the future that lies in that direction. State and county societies, here and there, have caught the meaning of and endorsed this program. But until the man in general practice is willing and determined to make the prevention of disease a real part of his everyday business, it will remain only a pleasant topic of conversation as far as he is concerned.

DISCUSSION (*Abstract*)

Dr. H. C. Ricks, Jackson, Miss.—We who have been actively engaged in the public health field during the last decade have had our minds in most instances completely changed concerning the relationship of the public health worker and his program to the private physician. A few years ago, the physician actively opposed the activities of the public health worker, not so much because of the program, but because the physician of the community where the program was being fostered had not been consulted or advised as to what was to be accomplished by the program and how. Many of us present have gone into communities and attempted to put on a program of preventive medicine without even calling upon the local physician. Therefore, he could be expected to oppose the work, for he knew nothing of our methods or for what reasons we were in the community. We have in the past overlooked the fact that the busy practitioner has not in many instances had the opportunity to know the latest methods of preventing disease. His medical literature does not carry information concerning disease prevention. The detail men from the manufacturers do not discuss preventive measures with him. Thanks to such leaders as Chapin of Providence, Rhode Island, Welch of Alabama, Williams of Virginia, and Leathers, now of Vanderbilt University, the public health worker today appreciates in the large majority of instances the place of the private physician in a public health program in a community, state or nation.

Our medical schools, which have until recent years taught only curative clinical medicine, are now giving their students real information concerning the field of preventive medicine. The Medical Departments of Vanderbilt University, University of Louisville, University of Tennessee and Tulane University are Southern schools maintaining departments of preventive medicine which are outstanding in this field.

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EDITORIAL DEPARTMENT

SOUTHERN MEDICAL ASSOCIATION
Twenty-Ninth Annual Meeting
St. Louis, Missouri, November 19-22, 1935

THE ST. LOUIS MEETING

The Southern Medical Association convenes in November in one of America's outstanding cities and medical centers. The two great medical colleges, Washington University School of Medicine and St. Louis University School of Medicine, the many well equipped hospitals and the distinguished profession make St. Louis a Mecca for physicians. For the first time in the history of the Association, a meeting is to be held in Missouri. St. Louis is accessible from all parts of the United States. It has unequalled railroad facilities, paved highways from north, south, east and west, and is on the leading air lines.

A modern municipal auditorium, the last word as a convention facility, is being completed. All meetings and all exhibits will be in this building, under one roof. Within convenient distance from the Auditorium is a sufficient number of first-class hotels to assure everyone of comfortable accommodations.

The first day, Tuesday, has been designated St. Louis Day. Clinical talks will be made by widely known physicians of St. Louis. Five clinical sessions will run concurrently, covering all the branches of medicine and surgery. On Wednesday forenoon there will be a similar program, conducted by physicians from other parts of the United States, from Massachusetts to California and from the Great Lakes to the Gulf of Mexico. Several foreign countries will be represented. All clinical sessions, five in number, Tuesday morning and afternoon and Wednesday morning, will be held in the Auditorium so that convention guests may go from one to another as they like without loss of time.

From Wednesday noon through Friday noon, the special sections working in half-day sessions will conduct their programs. These will provide material of more than usual interest arranged by able officers of sixteen sections of the Association. There will be, in addition, five conjoint meetings.

On Tuesday evening, there will be a general public session, and Wednesday evening the President, Dr. H. Marshall Taylor, will address a second general session, which will be followed by the President's reception and ball. Both evening meetings will be held in the large ballroom of the Jefferson Hotel.

On Thursday evening, the alumni reunion dinners are scheduled. A large attendance is expected, particularly by the alumni of the two St. Louis schools, Washington University and St. Louis University Schools of Medicine.

The host society, the St. Louis Medical Society, has a membership of one thousand and under the leadership of Dr. Neil S. Moore, its President, is leaving nothing undone which can be done for the success of the meeting. The St. Louis County Medical Society, with a membership of a hundred and fifty, under the leadership of Dr. R. L. Foster, its President, is cooperating heartily with the St. Louis Medical Society.

Now is the time to make plans for attending the most notable meeting of the year, in St. Louis, Tuesday, Wednesday, Thursday and Friday, November 19-22.

SCIENTIFIC EXHIBITS

The scientific exhibit is in many ways a more effective means of demonstrating a method or an idea than a paper or an address can be. The exhibits are a source of particular pride to the Association, and it would like to have in them each year a more complete record of advancing science in the South.

The St. Louis Auditorium provides an unusually adequate and convenient physical arrange-

ment for display of material. Physicians throughout the South who have something worth while to show are urged to apply for space as soon as possible by writing the Association office, Empire Building, Birmingham, Alabama.

STUDIES ON POLIOMYELITIS

In prevention of yellow fever, all methods of sanitation and antisepsis were useless until it was demonstrated that the infectious agent was transmitted by a single species of mosquito. In the case of poliomyelitis, an insect carrier has not been demonstrated, but attempts to infect monkeys have proven a remarkably limited path of invasion. The living body seems to be vulnerable to this virus at only two points, and these are smaller and more inaccessible than the heel of Achilles. Recent studies have brought out many facts of interest.

Infantile paralysis is said to resemble diphtheria in some of its reactions in the body, particularly in its choice of human victims.[1] Both are serious diseases of children, the susceptibility rate being lower than that of measles or whooping cough. With both, it is probable that light infections occur which gradually immunize a large part of the population. General health seems to bear no direct relation to susceptibility. Age is the potent factor, small children being more commonly susceptible, while the proportion of immunes increases with the age group. Probably most adults are immune. In the case of diphtheria, there is a test for susceptibility. In infantile paralysis, this practical aid is lacking.

Various biological fluids are reported to neutralize the virus of poliomyelitis outside the body. Among these are the nasal secretions of healthy adults, placental extract, pregnancy urine, vitamin C and several endocrine extracts. Undiluted human tears, according to Jungeblut,[2] of New York, rapidly inactivate it. Saliva and spinal fluid of the same individuals have no destructive effect, and spinal fluid even of monkeys convalescent from infantile paralysis and with a high titer of immune serum fails to neutralize the virus.

The pathology seems to be limited to the central nervous system, with no lesions outside the nervous tissues.[3] Flexner and Lewis showed some years ago that the path of infection is through the nose. Schultz and Gebhardt,[4] of Stanford University, in 1934, presented evidence that the virus after intranasal inoculation gains admission to the central nervous system through the nerves of smell. When these nerves were sectioned by cauterization of the olfactory bulbs, virus placed in the nose failed to induce the disease. The same animals were later infected by inoculation directly into the cerebrum, showing that the barrier was mechanical and local; they had no general immunity.

Lennette and Hudson,[5] of the University of Chicago, have confirmed and extended the observations of the Californians. Normal monkeys are extremely susceptible to intranasal inoculations of polio virus. To infect them by the intravenous route, massive doses must be used. Even then the virus is excreted from the blood stream into the nasopharynx and is present in the nasal washings before the onset of the disease. If the olfactory bulbs are cauterized, heavy intranasal inoculation does not produce the disease. After intravenous inoculation in the same animals, the virus may again be recovered from the nasal secretions, showing that it has been excreted into the nose, but the disease does not develop. Not only the usual entrance, but the sole entrance, in the monkey is apparently these nerves; even if the virus is in the circulating blood it can reach the susceptible central nervous system only through the olfactory passage.

Since these nerves are the only path of entry, elimination of them should close the door to systemic infection. Olfactory section thus possibly offers a surgical prevention of the disease. Any individual given the two alternatives, infantile paralysis or olfactory section, would sacrifice his sense of smell rather than his means of locomotion. In view of the uncertain course of infection even after definite exposure, nerve destruction will hardly be attempted as prophylaxis. A temporary harmless blockage of these vulnerable points by antisepsis during an epidemic would have experimental interest. In difficulty, it would probably compare with the difficulty of antisepsis in the blood stream. Though polio virus appears to be fairly easily recovered from nasal washings of monkeys with the disease, it

1. Zwemer, R. L.; and Jungeblut, C. W.: Effect of Various Cortico-adrenal Extracts on Diphtheria Toxin *in vivo* and *in vitro*. Proc. Soc. Exper. Biol. & Med., **32**:1583, June, 1933. Jungeblut, C. W.; and Steinbach, M. M.: idem, p. 1537.

2. Jungeblut, C. W.: Occurrence of Poliocidal Substances in Tears, Saliva and Cerebrospinal Fluid of Normal Individuals. Proc. Soc. Exper. Biol. & Med., **32**:1534, June, 1935.

3. Brodie, Maurice: Role of Convalescent Serum in Preparalytic Poliomyelitis. Jour. Immunol., **28**:353, 1935.

4. Schultz, E. W.; and Gebhardt, L. P.: Olfactory Tract and Poliomyelitis. Proc. Soc. Exper. Biol. & Med., **31**:728, 1934.

5. Lennette, E. H.; and Hudson, N. P.: Relation of Olfactory Tracts to Intravenous Route of Infection in Experimental Poliomyelitis. Ibid, **32**:1444, June. 1935.

is reported to be quite difficult to obtain it in the same manner from human beings.[1] This may indicate either a higher degree of human immunity, or a difference in the course of the infection.

GLEANINGS FROM RECENT JOURNALS

Gout.—Gout is accepted as a metabolic disease, usually with uric acid accorded the role of chief offender. In 1797, sodium urate crystals were identified in material from the tophi of gout. Later, an increase in the blood uric acid was shown. Talbott, Jacobson and Oberg[2] have made detailed studies of water and mineral metabolism upon two patients with gout, during attacks and at periods before and after. The attacks or cycles of the disease followed one another so closely that an indubitably normal period for these individuals could not be obtained.

In both patients it was noted that during the prodromal period of gout the urinary volume increased markedly, rising about 25 per cent above normal. This diuresis was accompanied by an increased total excretion of sodium and chlorine, as well as by increased concentrations of both these elements in the urine. There was thus a negative sodium and chlorine balance, or loss of those ions from the body. The excretion of phosphate, total nitrogen and urate was increased, but the urinary concentrations were not changed. The weight of the patient increased during an attack. During the recovery period, the urine volume was below normal for two days.

The metabolic disturbances of gout are thus apparently not limited to changes in uric acid, but are of a much more generalized nature, and include changes in water balance and in various electrolytes.

Prevention of Gangrene.—Thrombo-angiitis obliterans is a disease which is limited largely to men. Its pathology, according to McGrath,[3] of Cincinnati, closely resembles the pathology of ergot gangrene which may be induced in small animals, for example in the rat's tail, by injections of ergot extracts. In human epidemics of ergotism, it has been noted that gangrene does not occur in women. McGrath therefore undertook to learn whether experimental ergot gangrene might be affected by injections of estrin, the ovarian hormone.

The effects of subcutaneous injections of ergotamine tartrate upon a rat are described as follows: three to five days after administration of 25 to 100 milligrams per kilogram of body weight, the tail becomes pale and sensitive at the end. This is followed in six to nine days by cyanosis. The tail becomes very painful. Later it is black, dry, and anesthetic, and in sixteen to twenty days it sloughs off. McGrath administered ergot extract in varying doses to his rats. Twenty-four hours later theelin injections were begun and continued daily for a month. All animals which received ergot alone developed gangrene of the tail. All females receiving ergot and theelin remained in normal health without developing gangrene. In male rats, theelin had no preventive effect, but the tail sloughed off at the expected time. If sufficient theelin were given, ergot gangrene could always be prevented in the females. In some of the males which received theelin, there appeared to be a delay in the onset of symptoms. One wonders whether the male sex hormone comparable to theelin would have had any preventive effect in the male rats.

Recently it has been suggested that clinical thrombo-angiitis obliterans is one of the sequelae of an earlier attack of typhus fever. A small group of patients with the vascular disease gave a positive skin test for typhus.[4] Prevention of a particular type of experimental gangrene, ergot gangrene, by a specific medicant, theelin, is significant in the study of both the disease and the physiology of the hormone.

Abortifacients.—Tincture of iodine introduced directly into the uterus has been used to induce abortion for fifty years.[5] Since 1930, several irritant pastes have been advocated for the same purpose. "Interruptin" and "provocal" are described by D'Amour and Kiven,[5] of the University of Denver, as recently popular, particularly abroad. Because fatal accidents have followed their use, the effects were investigated upon the uterus of the rat.

1. Trask, J. D.; and Webster, L. T.: Isolation of Poliomyelitis Virus from the Nasopharynx. Jour. Exper. Med., **62**:245, Aug., 1935. Kramer, S. D.: Detection of a Healthy Carrier of Poliomyelitis without History of Contact. Proc. Soc. Exper. Biol. & Med., **32**:1165, 1935.

2. Talbott, J. H.; Jacobson, B. M.; and Oberg, S. A.: Electrolyte Balance in Acute Gout. Jour. Clin. Investig., **14**:411, July, 1935.

3. McGrath, E. J. G.: Experimental Peripheral Gangrene: Effect of Estrogenic Substance. Relation to Thrombo-angiitis Obliterans. Arch. Int. Med., **55**:942, June, 1935.

4. Goodman, Charles: A Skin Test to Suggest the Diagnosis of Recovered Typhus and Thrombo-angiitis. Bull. N. Y. Acad. Med., **11**:403-412, June, 1935. Goodman, C.; and Brodie, Maurice: A Test for Diaganosis of Thrombo-angiitis Obliterans. Proc. Soc. Exper. Biol. & Med., **32**:1331, 1935.

5. D'Amour, F. E.; and Kiven, Nathan: Harmful Effects of Certain Chemicals upon the Uterus of the Rat. Amer. Jour. Obst. & Gyn., **29**:503, April, 1935.

Leunbach's paste ("provocal"), say the Denver workers, contains soap, iodine, potassium iodide, tincture of benzoin, myrrh and thymol. The composition of "interruptin" is very similar. When Leunbach's was introduced in full strength into the uterus of pregnant rats, it invariably terminated the pregnancy. However, a number of the animals died, and autopsy showed infection of the entire abdominal cavity. The uterus was practically destroyed in many. The animals which survived injections of diluted paste were bred a few months later, but few or no conceptions occurred.

If sterile solutions of such substances as physiological potassium or sodium chloride were injected into the gravid uterus, abortion often followed. The different ingredients of Leunbach's paste were administered separately to groups of animals to determine the effects of each. Neutral soap in a concentration of one to ten caused abortion, and the appearance of the uterus at the second attempted pregnancy was about like that of the animals which had been injected with the whole paste. Iodine alone induced abortion, with similar damaging effects. Several alkaline, hypertonic and antiseptic solutions were studied. All which regularly induced abortion so injured the uterus that breeding three months later was not followed by normal pregnancy.

Endometrial Hyperplasia.—In attempted clinical use of the ovarian follicle hormone, estrin, enormous doses are sometimes advocated. Estrin induces rutting and vaginal changes in small animals, with changes in the endometrium of the uterus, rutting being usually taken as the sign of an effective dose or active product. Simpson and Burch,[1] of Nashville, show that quantities of the hormone much too small to induce vaginal changes will cause hyperplasia of the lining of the uterus of the guinea pig.

Conceivably, estrin given to women may alter the endometrium without other discernible effects; and small excesses of estrin in the body may result in the enormous proliferation of the endometrium which is observed in certain patients.

1. Simpson, John W.; and Burch, John C.: Experimental Production of Glandular Cystic Hyperplasia of the Endometrium with Estrogenic Substance of Parturient Urine. Proc. Soc. Exper. Biol. & Med., **32**:1570, June, 1935.

TWENTY-FIVE YEARS AGO

FROM JOURNALS OF 1910

Transmission of Poliomyelitis.[1]—"Flexner and Lewis * * * have issued a preliminary report of their experiments in the inoculation of monkeys with the material obtained from two cases dying of acute poliomyelitis. An emulsion in salt solution of the spinal cord from the children * * * was introduced through a trephine opening in the skull. In each instance the inoculated animal became paralyzed after a somewhat variable period. Histological study of the spinal cords of the monkeys showed in every case lesions similar to those of poliomyelitis in man.

"[It is difficult to overestimate the importance of this communication. The power of reproducing the disease in an indefinite series of animals may well prove to be the first step in the production of a curative or preventive serum] W. S. Thayer."

Heart Massage in Surgery.[2]—White * * * says that heart massage is an established method of resuscitation, ten successful cases having been reported by ten operators. * * * The best results have been obtained by the subdiaphragmatic method. * * * The possibility of resuscitation bears a definite relation to the time that has elapsed between the cessation of the heart beat and massage. The briefer the interval the more rapid is the response to heart massage."

Obesity.[3]—Laissus, * * * after a study of 200 cases of obesity, recommends as of primary importance a reduction in the amount of carbohydrates and fat ingested. * * * Massage, to be beneficial, should be prolonged for some time, and the kneading process is preferable."

Treatment of Pertussis.[4]—"Soldin * * * says that eucalyptus leaves have been advocated for a long time in the treatment of various respiratory diseases. * * * Soldin made use of two commercial preparations * * * in the treatment of pertussis * * * it lessened the number and severity of paroxysms in most of the cases. * * * He advocated its use because it may be given with perfect safety."

Cause of Pernicious Anemia.[5]—"Berger and Tsachiya * * * extracted with ether the mucous membrane of the gastro-intestinal tract of cases of pernicious anemia after death and obtained a lipoid substance which in vitro as well as experimentally on animals had a marked hemolytic action. The controls made with normal mucous membrane showed only a slight hemolytic action. They then produced a catarrhal inflammation of the gastric mucous membrane of dogs. The ether extracts of the involved mucous membrane contained a similar lipoid body. From these experiments they draw the conclusion that the cause of pernicious anemia is a chronic catarrh of the mucous membrane of the gastro-intestinal tract and the consequent production of a hemolytic substance. They explain the beneficial effects of intestinal lavage and diet in pernicious anemia upon this same etiological hypothesis."

1. Amer. Jour. Med. Sci., **139**:132, 1910.
2. Surg. Abst., Amer. Jour. Med. Sci., **139**:137, 1910.
3. Therapeutics. Ibid, p. 141.
4. Pediatrics. Ibid, p. 298.
5. Therapeutics. Ibid, p. 611.

St. Louis—Where We Meet

MEDICAL ST. LOUIS*

St. Louis has attained remarkable commercial prominence, but in pursuing this commercial success it has not neglected intellectual development. It is today one of the truly outstanding medical centers of this country and is rapidly progressing toward international prominence. It presents unusual facilities for the care of the sick, for research into the cause and cure of disease, and for the instruction of medical students. St. Louis is proud of its two great medical schools.

WASHINGTON UNIVERSITY SCHOOL OF MEDICINE

St. Louis Medical College, founded in 1842, was admitted as a department of the University in 1891. In 1899 the Missouri Medical College, which was founded in 1840, was united with the St. Louis Medical College to form the medical school of Washington University.

Washington University School of Medicine, Central Group (Barnes Hospital).

This school therefore continues the work of the St. Louis Medical College and the Missouri Medical College, two of the oldest medical schools west of the Mississippi River.

In 1910, the corporation of the University, appreciating the valuable service which a medical school can render to the community, with the cooperation of the medical faculty, reorganized the school in all departments and appointed heads of departments and instructors in anatomy, physiology, biological chemistry, pathology, medicine, surgery and pediatrics, who devote themselves to teaching and research, and associated with this staff clinical instructors chosen from the medical profession of St. Louis.

In 1914 the Washington University School of Medicine moved from its old location on the corner of Eighteenth and Locust Streets to the new buildings facing Forest Park on the corner of Kingshighway Boulevard and Euclid Avenue. The buildings of the School of Medicine form a part of a medical group known as the Barnes Hospital group.

The affiliation of Barnes Hospital, St. Louis Children's Hospital, McMillan Eye, Ear, Nose and Throat Hospital and Oscar Johnson Institute with Washington University School of Medicine makes these institutions for teaching purposes integral parts of the School of Medicine.

The Washington University Dispensary serves as an out-patient clinic for the University group of hospitals.

Further teaching facilities are afforded by the St. Louis City Hospital, including the Isolation Hospital and the City Sanitarium.

An excellent library is housed in the medical school and several museums and collections for teaching ond investigation are maintained by the various departments. Recently a valuable collection of anatomical specimens prepared with unusual skill and care by Bohumil Hochmann has been purchased for the museum. Diseases of bones are well illustrated by a large number of specimens collcted during the early history of the school by Dr. Charles A. Pope and Dr. John T. Hodgen. A section of the museum contains experimental pathological lesions.

The Beaumont Room, adjoining the main reading room of the library, contains manuscripts, letters and other valuable material of the pioneer American physiologist, William Beaumont, presented to the University by his granddaughter, the late Lily Beaumont Irwin.

ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE

The school of medicine of St. Louis University was established in 1837 in a small house on Washington Avenue between Tenth and Eleventh Streets. In 1847 the school was moved to its own college building on Seventh and Clark Avenues. The affiliation between the medical school and the University was severed in 1855, and the medical department became known as the St. Louis Medical College and functioned as an independent school.

The Marion Sims College of Medicine, founded in 1890 and occupying a building erected for the purpose on Grand Boulevard and Caroline Street, combined in 1901 with the Beaumont Medical College, organized in 1886, and became known as Marion Sims-Beaumont Medical College. In 1903 the University assumed control of these schools, which had been merged two years previously, and became known as the St. Louis University School of Medicine. In the reorganization which ensued the fundamental departments, anatomy, chemistry, physiology, pathology, bacteriology and pharmacology, were placed under the direction of full-time departmental heads.

The main building of the original Marion Sims Col-

*By Dr. R. V. Powell, St. Louis Chairman of Publicity for Southern Medical Association meeting.

St. Louis University School of Medicine, Main Building (Administration, Etc.). Picture taken through front entrance of University (Firmin Desloge) Hospital, just across the street.

lege of Medicine was used until 1927, when the present medical buildings were erected. The buildings are located on Compton Hill, one of the highest points in the City of St. Louis, the main building facing Grand Boulevard at Caroline Street.

An excellent medical library, which has been enriched by several private collections, is housed in the south wing of the building.

The University Hospital, affording clinical teaching facilities to the Medical School, embraces a group of three hospitals: the Firmin Desloge Hospital, St. Mary's Hospital and Mt. St. Rose Sanitarium. The Sisters of St. Mary have placed their educational and medical activities in these institutions under the complete control of the University. By an agreement between the Sisters and the University the Directors of the various departments of the school of medicine are made responsible for the corresponding hospital departments, thus forming a compact organization, assuring the closest university supervision and the highest type of medical, educational and nursing efficiency.

The Alexian Brothers', St. Anthony's and St. John's Hospitals are known as the "Associated Hospitals." These hospitals afford further teaching facilities and in return are given leadership and guidance in their medical and educational activities. The staffs are appointed by the University.

Intimate association is also maintained between the St. Ann's Lying-In Infirmary, St. Ann's Foundling Society and with the St. Louis Obstetric Dispensary. These institutions are utilized as teaching centers for obstetrics and pediatrics. Opportunity is also afforded for teaching in the St. Louis City Hospital, Isolation Hospital and St. Louis City Sanitarium.

INDEPENDENT HOSPITALS

The hospitals which are directly connected with the two schools of medicine have already been briefly mentioned. There are numerous other excellent hospitals in St. Louis worthy of mention, but space does not permit going into details concerning their many prominent features. Among these independent hospitals are the Barnard Free Skin and Cancer Hospital, Bethesda Hospital, Christian Hospital, DePaul Hospital, Evangelical Deaconess' Home and Hospital, the Frisco Employees' Hospital, Jewish Hospital, Lutheran Hospital, Missouri Baptist Hospital, Missouri Pacific Hospital, St. Ann's Widows' Hospital, St. Louis City Hospital, No. 1, St. Louis County Hospital, St. Luke's Hospital, Shriners' Hospital and United States Marine Hospital.

St. Louis' medical schools and hospitals will be of outstanding interest to visitors.

Book Reviews

Clinical Laboratory Methods and Diagnosis: A Textbook on Laboratory Procedures with Their Interpretation. By R. B. H. Gradwohl, M.D., Director of the Gradwohl Laboratories; Director of the Gradwohl School of Laboratory Technic; Director of Laboratories, St. Louis County Hospital; Chief of Staff and Pathologist to Christian Hospital; Lieutenant Commander, Medical Corps, Fleet, United States Naval Hospital. 1028 pages, illustrated. St. Louis: The C. V. Mosby Company, 1935. Cloth, $8.50.

This is a very reliable compilation of laboratory tests and pathologic procedures. The technics are those which are considered standard for urinalysis, blood chemistry, hematology, gastric analysis, puncture fluids, sputum, feces, parasitology, bacteriology, serology, basal metabolism, postmortem examinations, tissues, preparation of museum specimens, and toxicologic technic. Clinical interpretations of the tests are included. Hematology has received special emphasis and the methods and interpretations of Schilling are given in detail. Bacteriology and parasitology are discussed with more than usual completeness for a text of this nature. The section on postmortem technic will be welcomed for its explicit directions. Dr. Gradwohl has greatly expanded this volume and produced a comprehensive work. It is well illustrated and will prove of value to clinician, student and laboratory worker.

The International Medical Annual, a Year Book of Treatment and Practitioner's Index. By, Editors: H. Letheby Tidy, M.A., M.D., Oxon, F.R.C.P., and A. Rendle Short, M.D., B.S., B.SC., F.R.C.S. 522 pages, illustrated. Baltimore: William Wood & Co., 1935. Cloth, $6.00.

These short articles on the outstanding medical and surgical advances of the past year include abstracts from the current literature and also from new books. Among the interesting reviews are those on toxemias as a cause of psychoses, the importance of childhood psychoses as related to the adult type, the St. Louis en-

cephalitis epidemic, carcinoma of the lung, lobectomy, unpadded plaster casts, thyroid heart, the barbiturates, and the danger signals of coming eclampsia. The method of presentation, the numerous plates and illustrations and the excellence of the paper and binding as well as the subject matter, all lend themselves to make this a desirable volume.

Textbook of Urology for Students and Practitioners. By Daniel N. Eisendrath, M.D., Consulting Urologist to the American Hospital, Paris, France; Assistant Professor of Surgery (Genito-Urinary) Rush Medical College of the University of Chicago, and Harry C. Rolnick, M.D., Clinical Professor of Genito-Urinary Diseases, Loyola University Medical School. 942 pages, illustrated. Philadelphia: J. B. Lippincott Company, 1934. Cloth, $10.00.

The book begins with short chapters on embryology, anatomy and physiology of the genito-urinary organs. A short but valuable chapter is included on urography and one on anesthesia in urologic procedures. Venereal diseases are given more space, detail and clarity than in the usual work of this kind. The treatment of acute Neisserian infections as outlined is more energetic than that approved by most urologists. Treatment is recommended every one to two hours in the first few days of urethritis in the male. The prognosis in secondary lues is expressed as follows: "With the development of frank secondary syphilis, treatment, although it may control the infection, rarely cures the patient."

The various anomalies, injuries, affections and infections are discussed as their relative importance indicates. The operative technic for various minor and major urologic operations is given. With very few exceptions, the book seems to be in accord with the generally accepted modern urologic ideas. The bold type presentation of the most important matter makes it unusually valuable for students as well as for reference. It is a book which anyone interested in urology will be fortunate to own.

Doctors and Juries: The Essentials of Medical Jurisprudence. By Humphreys Springstun, of the Detroit Bar. 155 pages. Philadelphia: P. Blakiston's Son & Co., Inc., 1935. Cloth, $2.00.

The every-day relations between doctor and patient and doctor and law are covered from a legal standpoint and after reading the book the physician reaches the conclusion that he has some legal rights, but that they are rather limited.

The book is readable and enlightening, but not instructive.

The Technique of Postmortem Examination as Practiced in the Pathological Institute of McGill University at the Royal Victoria Hospital, Montreal. Compiled by D. R. Coman, M.D., C.M., Assistant to the Institute and Demonstrator in Pathological Anatomy. 47 pages, illustrated. Montreal: Renouf Publishing Company, 1934. Cloth, $1.40.

No one will deny the value of the lessons to be learned at the autopsy table, yet few outside the larger centers make any serious attempt to check their clinical observations by frequent postmortem study. In as far as such an attitude results from a feeling of unfamiliarity with autopsy technic, the present volume should prove of value far beyond its modest size. The style is clear and direct, no time is wasted on non-essentials and the whole book can be read in an hour. It should encourage a wider application of that postmortem study without which no physician can expect to attain his normal growth in diagnostic skill and the fundamental understanding of disease processes.

Injuries and Their Treatment. By W. Eldon Tucker, M.A., B.Ch. (Cantab.), F.R.C.S. (Eng.), Surgeon to St. John's Hospital, Lewisham; Surgeon to the London Clinic for Injuries, Baker Street; Late Registrar Royal National Orthopedic Hospital. 173 pages, illustrated. New York: Oxford University Press, 1935. Cloth, $3.75.

The text of this volume is the judicious use of exercise and manipulation in injuries. Fractures are dealt with only in certain cases. The author stresses the fact that it is not a book on the treatment of fractures. Its scope embraces the pathology of soft structure injuries, and the author stresses the importance of early motion, massage, the aspiration of joints with effusion, the massaging and breaking up of hematomas, either intramuscular or about the joints.

The methods of active treatment of a joint according to the author are divided into three main groups:

(1) Movements, which are variously subdivided; (2) application of heat (radiant, infra-red, diathermy and couterirritants; and (3) massage, which if applied gently will assist the damaged circulation without doing further injury.

Warning is given that the manipulation of joints should never be excessive. Manipulation under anesthesia is not used unless the other types of manipulation have failed, and even then, it is never carried out with such vigor as to produce excessive reaction.

Repeated warnings are given in that every injury, especially joint injury, should be x-rayed before it is manipulated.

The suggestions regarding massage of hematomas and dispersing the hemorrhage into the surrounding normal tissue, especially about the ankle joint, have not been entirely accepted by the majority of authorities.

The volume is excellent in the hands of surgeons who are doing a large amount of traumatic work. It may cause the over-enthusiast or faddist to become too active and inconsiderate in carrying out rigid massage and passive motions. It should be a supplement for the experienced surgeon, not the major source of information for the inexperienced.

Medicine Marches On. By Edward Podolsky, M.D. 343 pages. New York: Harper & Brothers, 1934. Cloth, $3.50.

This book appeals to the dramatic and the sensational. While it records many facts, it includes some unproved hypotheses. There is a tendency to color the whole book with a rosy hue which will give the layman an exalted idea of modern medicine and modern practitioners, and which may lead uninformed persons into disappointments.

Aids to Ophthalmology. By N. Bishop Harman, M.A., M.B. (Cantab.), F.R.C.S. (Eng.), Hon. LL.D. (Man.), Consulting Ophthalmic Surgeon, West London Hospital; Lecturer in Anatomy, King's and Caius Colleges, Cambridge. 242 pages, illustrated. Baltimore: William Wood & Co., 1935. Cloth, $1.25.

The writer assumes that the student is familiar with the anatomy of the eye. He gives a condensed account of the commoner ophthalmological conditions and their treatment. There are also several chapters on refraction. A chapter on "Vision Standards," that is of little value to the American student, and thirty-eight new illustations have been added. This is a satisfactory compend.

Southern Medical News

THE COLUMBIA MEETING

The Boone County Medical Society will be host to the annual meeting of the Ninth Councilor District of the Missouri State Medical Association at Columbia, Missouri, Tuesday, September 17. This meeting is planned as a *Southern Medical Association Day* in Central Missouri. The principal part of the program is to be put on by men who are and have been actively connected with the Southern Medical Association. The meetings will be held at the Columbia Country Club and the program will begin at 3:00 p. m. and will be concluded at a dinner that evening.

Dr. Frank K. Boland, Atlanta, Georgia, Professor of Clinical Surgery, Emory University School of Medicine, will present the subject "Surgical Treatment of Pulmonary Tuberculosis." Dr. Boland has been active in the Southern Medical Association for many years, serving the constitutional time as a member of the Council and last year as its Chairman.

Dr. Seale Harris, Birmingham, Alabama, one time Professor of Medicine, University of Alabama School of Medicine, will present the subject "The Diagnosis and Treatment of Hyperinsulinism" (Lantern Slides). Dr. Harris served the Southern Medical Association as Secretary-Treasurer and Editor for many years, as President and as a member of the Board of Trustees, last year as its Chairman.

Dr. Quitman U. Newell, St. Louis, Associate Professor of Obsterics and Gynecology, Washington University School of Medicine, will present the subject "The Importance of the Recognition of Early Uterine Carcinoma with Some Remarks on Treatment" (Lantern Slides). Dr. Newell is the General Chairman for the St. Louis meeting of the Southern Medical Association, being appointed to that position by the President of the host society, the St. Louis Medical Society.

Mr. C. P. Loranz, Birmingham, Alabama, Secretary, Treasurer and General Manager of the Southern Medical Association, will present the subject "Some Aims of the Southern Medical Association and a Pre-View of the St. Louis Meeting."

Dr. W. P. Dysart, Columbia, is President of the Boone County Medical Society, and Dr. A. R. McComas, Sturgeon, is the Councilor of the Ninth Councilor District. The Committee on Speakers and Invitations is: Dr. Frank G. Nifong, Dr. Dudley S. Conley and Dr. Dudley A. Robnett, of Columbia. The Committee on Program and Arrangements is: Dr. M. Pinson Neal, Dr. Carl M. Sneed, Dr. Maurice E. Cooper, Dr. E. D. Baskett and Dr. Stephen D. Smith, of Columbia.

The members of the Missouri State Medical Association are cordially invited to this District meeting. Columbia is accessible from all over the State, but particularly so from Central Missouri.

ALABAMA

Dr. H. Earle Conwell, Birmingham, was guest speaker before the Franklin County Medical Society at their meeting in Russellville July 2.

Dr. Judson D. Dowling and Miss Fleta McWhorter, both of Birmingham, were married June 8.

Deaths

Dr. James David Jones, Sweetwater, aged 68, died May 24 of angina pectoris.

Dr. John Henry Walling, Vinemont, aged 85, died April 15 of chronic myocarditis.

ARKANSAS

Dr. Gordon Hastings, Little Rock, has returned after completing an eight months' course in public health administration at Harvard University and has resumed his duties as Assistant State Health Officer.

Dr. H. A. Murphy, El Dorado, recently opened a maternity hospital.

Dr. L. J. Kosminsky, Texarkana, was a recent guest speaker before the Ouachita Parish Medical Society at Monroe, Louisiana.

Dr. J. M. Hooper, Batesville, has been made Lieutenant-Colonel in the Medical Reserve Corps, U. S. A.

Dr. J. A. Foltz, Fort Smith, was a speaker before the annual banquet session of the Tulane Alumni Association in New Orleans on June 7.

Dr. William Hibbitts, Texarkana, has been made Chief Surgeon of the Cotton Belt Lines.

Dr. A. J. Hamilton was recently appointed Health Officer at Rison.

Dr. G. L. Hardgrave, Clarksville, is the new Vice-President of the University of Arkansas Alumni Association.

Dr. C. H. Dickerson, Conway, has been elected President of the Country Club of that city.

Dr. N. B. Daniel, formerly of Texarkana, has moved to Mercedes, Texas.

Dr. H. H. Holt, Nashville, and Miss Elizabeth Abbie Seale were married July 18.

Deaths

Dr. Irvin Shepherd, Sheridan, aged 67, died June 16 of encephalitis.

Dr. John D. Carter, Grapevine, aged 56, died recently of pneumonia.

Dr. John G. Love, Hartman, aged 56, died July 14.

Dr. J. P. Sheppard, Little Rock, aged 60, died July 21 of injuries received in an automobile accident.

DISTRICT OF COLUMBIA

Major General Charles R. Reynolds on June 1 succeeded Major General Robert U. Patterson as Surgeon General of the United States Army.

Dr. William Thornwall Davis, Washington, was guest speaker before the Tennessee Valley Medical Association and Postgraduate Assembly at its meeting in Knoxville, Tennessee, in June.

FLORIDA

The Florida Society of Dermatology and Syphilology elected the following officers at their regular quarterly meeting held in Jacksonville June 29: Dr. J. Lee Kirby-Smith, Jacksonville, President; Dr. Wiley M. Sams, Miami, Secretary.

The Florida State Radiological Society held its annual meeting at Ocala May 12.

The Orange County Medical Society held its annual picnic and barbecue at Lakeside Park, Orlando, July 27.

Dr. J. H. Lucinian, Miami, is spending several weeks in the X-Ray Department of the Mayo Clinic after visiting x-ray clinics in Chicago.

Dr. James L. Estes, Tampa, has returned from Philadelphia, where he did some work at the Urological Clinic of the Pennsylvania Hospital.

Dr. J. A. Pires, Orlando, has returned from New York, where he took postgraduate work in roentgenology.

Dr. Walter A. Weed, of Orlando and Lakeland, spent two weeks studying special phases of roentgenology in Washington and New York recently.

Dr. Spencer A. Folsom, Orlando, has returned after spending six weeks in Pittsburgh at the Mercy Hospital studying electrocardiography.

Dr. Frank Gray, Orlando, has returned from Rochester, where he spent some time in the Surgical Division of the Mayo Clinic.

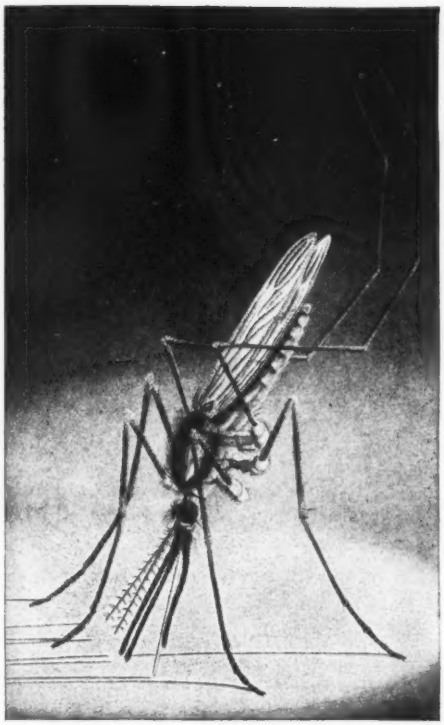
Dr. Ralph F. Allen, Milton, and Miss Eugenia Dillon were married June 17.

Dr. Clyde O. Anderson, St. Petersburg, and Miss Eileen Masters were married June 20.

Deaths

Dr. Paul Colson Perry, Jacksonville, aged 60, died June 5 of pulmonary embolism following an operation.

Continued on page 20



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421 M

Continued from page 866

Dr. Edward Porter Webb, Crestview, aged 63, died May 26.

Dr. Abraham Daniel Murphey, Fort Pierce, aged 71, died June 1 of septicemia and endocarditis.

GEORGIA

Dr. George Lombard Kelly, Vice-Dean and Professor of Anatomy, University of Georgia School of Medicine, Augusta, was recently elected Dean, effective September 1.

Dr. Roy R. Kracke, Emory University, at the recent meeting of the American Society of Clinical Pathologists, was elected President-Elect.

The Georgia Pediatric Society has appointed the following Scientific Committee: Dr. W. L. Funkhouser, Atlanta, Chairman; Dr. M. Hines Roberts, Atlanta; Dr. Joseph Yampolsky, Atlanta.

Dr. William S. Goldsmith, Atlanta, has been elected President of the Alumni Association of the Medical Department of Emory University School of Medicine.

Dr. Samuel P. Hall, Jr., formerly of Chickamauga, has been named Walker County Health Commissioner, succeeding Dr. Rufus F. Payne, resigned.

Dr. Joe B. Peniston, Newnan, on June 20 entertained the members of the Coweta County Medical Society with their families at a barbecue at Wynn's Pond.

Dr. James E. Paullin, Atlanta, President of the Georgia Medical Association, was a guest speaker before the Tennessee Valley Medical Association at Knoxville, Tennessee, in June.

Dr. Abe Davis, Americus, after taking postgraduate work at Vanderbilt University School of Medicine, Nashville, Tennessee, has resumed his duties as Sumter County Commissioner of Health.

Dr. J. D. Applewhite, Macon, has resumed his duties as Macon Health Officer after taking postgraduate work at Harvard University Medical School, Boston.

Dr. J. R. Sams, Covington, is now located in the Turner Memorial Building.

Dr. Julian Quattlebaum, Savannah, has returned after spending some time in Rochester in the Surgical Division of the Mayo Clinic.

Dr. D. H. Garrison, formerly of Tate, has located in Clarksville, where he will be associated with his brother, Dr. W. H. Garrison.

Dr. Tom Vann Willis, Brunswick, and Miss Ouida Jayne Temples were married June 8.

Dr. Thomas Oscar Vinson, Macon, and Miss Roesel Stanford were married June 6.

DEATHS

Dr. Robert McGehee Exley, Pineora, aged 48, died recently of cerebral hemorrhage.

Dr. Henry G. Maxey, Woodville, aged 43, died June 14 of furuncle and erysipelas.

Dr. James A. Lester, Fayetteville, aged 65, died June 24 of angina pectoris.

Dr. Edward Pinson McLennan, Parrott, aged 58, died May 31.

Dr. Elijah A. Lambert, Denton, aged 60, died June 21.

Dr. Alton R. Danforth, Norcross, died June 5.

KENTUCKY

The annual meeting of the Kentucky State Medical Association will be held September 30, October 1, 2 and 3 at Louisville.

Dr. Frank H. Mayfield, formerly of Richmond, Virginia, has located in the Brown Building, Louisville.

Dr. Philip E. Blackerby, Jr., Louisville, and Miss Clara Mae Hartmetz were married June 1.

DEATHS

Dr. Thomas A. Wash, Harrodsburg, aged 56, died June 12 of chronic myocarditis and arteriosclerosis.

Dr. John Henry Rice, Hopkinsville, aged 64, died June 7 of gangrene of the left hand and anemia.

Dr. Benjamin F. Owens, Louisville, aged 92, died recently of cerebral hemorrhage.

Dr. Walter Cox, Irvine, aged 46, died June 6 of pneumonia and miliary tuberculosis.

Dr. J. Stuart Wallingford, Paris, aged 66, died recently of angina pectoris.

Continued on page 22

DOCTOR! YOU ARE INVITED TO ATTEND

THE OKLAHOMA CITY CLINICAL SOCIETY'S

SIXTH ANNUAL FALL CLINICAL CONFERENCE

November 4, 5, 6, 7, 1935

Sixteen Distinguished Guest Lecturers

DR. FRED LYMAN ADAIR, Obs. & Gyn., Chicago, Head of Dept. Obs. & Gyn., Chicago Lying-In Hospital

DR. HARRY L. BAUM, Oto-Laryngology, Denver, Staff of Presbyterian, Childrens, and Denver General Hospitals

DR. BARNEY BROOKS, Surgery, Nashville, Prof. of Surg., Vanderbilt University Hospital

DR. JAMES T. CASE, Radiology, Chicago, Prof. of Radiology, Northwestern Univ. Med. School

DR. JOHN R. CAULK, Urology, St. Louis, Prof. of Clin. G-U Surg., Washington Univ. Sch. of Med.

DR. MAX CUTLER, Surgery, Chicago, Tumor Clinic, Michael Reese Hospital

DR. PALMER FINDLEY, Gynecology, Omaha, Attending Gyn., Swedish Mission & Methodist Hosps.

DR. FREDERICK J. GAENSLEN, Orthopedics, Milwaukee, Prof. of Orth. Surg., Univ. of Wisconsin Med. School

DR. CLIFFORD G. GRULEE, Pediatrics, Chicago, Prof. Pediatrics, Rush Med. College, Univ. of Chicago

DR. RUSSELL L. HADEN, Internal Medicine, Cleveland, Chief of Medicine, Cleveland Clinic

DR. JAMES S. McLESTER, Internal Medicine, Birmingham, President American Medical Association

DR. GRIER T. MILLER, Internal Medicine, Philadelphia, Asst. Prof. of Med., Univ. of Pa. Sch. of Med.

DR. C. S. O'BRIEN, Ophthalmology, Iowa City, Prof. of Ophthal., Univ. of Iowa Sch. of Med.

DR. PAUL A. O'LEARY, Dermatology, Rochester, Head of Dept. of Derm. & Syph., Mayo Clinic

DR. FRED W. RANKIN, Surgery, Lexington, Former Prof. of Surg., Univ. of Louisville

DR. RALPH M. WATERS, Anesthesia, Madison, Prof. of Anesthesia, Univ. of Wisconsin Med. Sch.

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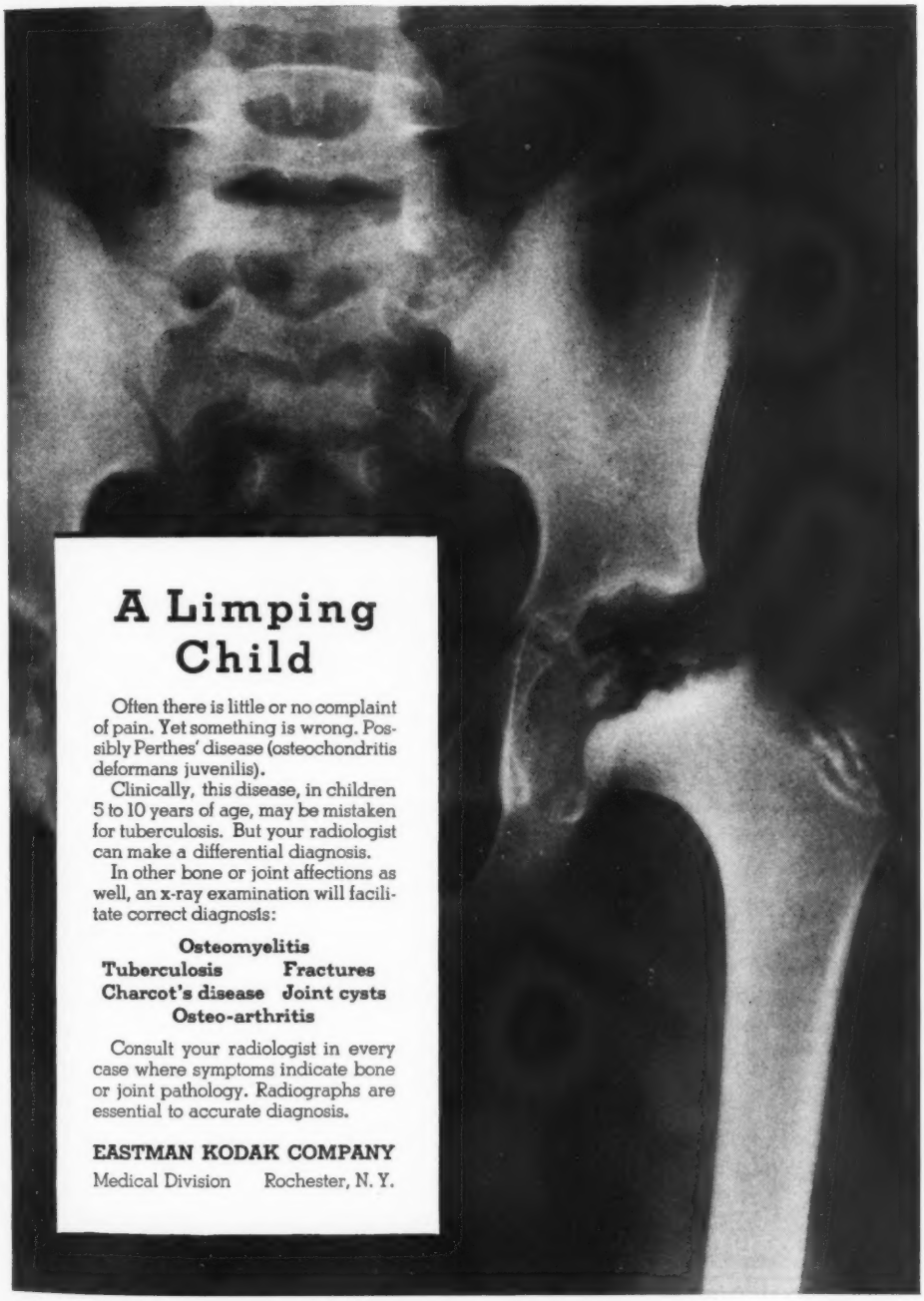
A Limping Child
Often there is little or no complaint of pain. Yet something is wrong. Possibly Perthes' disease (osteochondritis deformans juvenilis).
Clinically, this disease, in children 5 to 10 years of age, may be mistaken for tuberculosis. But your radiologist can make a differential diagnosis.
In other bone or joint affections as well, an x-ray examination will facilitate correct diagnosis:
Osteomyelitis
Tuberculosis Fractures
Charcot's disease Joint cysts
Osteo-arthritis
Consult your radiologist in every case where symptoms indicate bone or joint pathology. Radiographs are essential to accurate diagnosis.
EASTMAN KODAK COMPANY
Medical Division Rochester, N. Y.

Radiographs Provide Diagnostic Facts

Continued from page 20

Dr. David Howard McKinley, Winchester, aged 50, died May 21.

Dr. Cassius Dudley Mansfield, Stanton, aged 66, died May 26 of heart disease.

LOUISIANA

Dr. J. A. Foltz, Fort Smith, Arkansas, was a speaker before the annual banquet session of the Tulane Alumni Association on June 7.

Dr. L. J. Kosminsky, Texarkana, Arkansas, was a recent guest speaker before the Ouachita Parish Medical Society at Monroe.

Deaths

Dr. Joseph S. Martin, St. Martinsville, aged 62, died May 15.

MARYLAND

Dr. George O. Sharret, Cumberland, has been elected a member of the Board of Medical Examiners of Maryland to fill the unexpired term of the late Dr. Henry M. Fitzhugh, Westminster.

Deaths

Dr. Benjamin S. Warren, Chevy Chase, aged 63, died recently of cerebral hemorrhage.

Dr. James A. Stevens, Easton, aged 81, died June 8 of endarteritis obliterans and gangrene of the left foot.

Dr. John E. Thomas, Baltimore, aged 70, died recently of cerebral hemorrhage.

Dr. James Armitage Emery, Chevy Chase, aged 68, died July 28 of a heart attack.

MISSISSIPPI

Dr. A. P. Alexander, Como, has been appointed Health Officer of Panola County.

Dr. H. B. Watkins, Noxapater, has been appointed Health Officer of Winston County.

Dr. B. B. Martin, Jr., has located in Vicksburg for the practice of medicine, where he will be associated with his father at the Vicksburg Infirmary.

Dr. L. B. Otken, Greenwood, has been elected Commander of the American Legion of that city.

The Northeast Mississippi Thirteen Counties Medical Society had as guest speakers at a recent meeting Dr. J. A. Crisler, Sr., and Dr. Harley Harris, Memphis, Tennessee, and Dr. Walter Friday, Panama, Canal Zone.

Deaths

Dr. John Hillman McLain, Jackson, aged 47, died recently.

Dr. S. E. Rees, Purvis, aged 64, died recently.

MISSOURI

The American Hospital Association will hold its annual meeting in St. Louis September 30 to October 3.

Dr. M. P. Ravenel, Columbia, has been elected an honorary member of the Board of Directors of the National Tuberculosis Association.

Dr. W. J. Bryan, Mount Vernon, has been elected an active member of the Board of Directors of the National Tuberculosis Association.

Dr. Ralph S. Muckenfuss, St. Louis, has accepted a temporary appointment, pending an open civil service examination, as Associate Director of the Municipal Bureau of Laboratories of the New York Health Department, succeeding Dr. William H. Park, retired.

Dr. Quitman U. Newell, St. Louis, was a guest speaker before the Miami County Medical Society at Peru, Indiana, in June.

Dr. F. D. Dorsey, Keokuk, Iowa, was a guest speaker before the Caldwell-Livingston County Medical Society.

Deaths

Dr. William Emile Cramer, Kansas City, aged 70, died May 31 of perforation of the gallbladder.

Dr. Wallace Ainger Armour, Kansas City, aged 67, died June 14 of cerebral hemorrhage.

Continued on page 26

Dilaudid

(dihydromorphinone hydrochloride)

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DOSE:

Oral, subcutaneous and rectal.

For Pain: 1/48 to 1/16 grain

For Cough: 1/64 to 1/32 grain

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Dilaudid is made in U. S. A.

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In Surgery - - Pre-operatively, as an adjunct to anesthesia and post-operatively. Less likely to cause nausea or constipation than morphine; a stronger analgesic with a less pronounced soporific action.

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THE INCREASED NEED for calcium during pregnancy often necessitates the use of a dietary supplement which will provide an adequate amount of this element. Dicalcium Phosphate Compound with Viosterol Squibb not only supplies calcium, but enough phosphorus to maintain a proper balance of these minerals together with sufficient Vitamin D to ensure their utilization. Thus more satisfactory results may be expected than with calcium alone.

Each tablet contains 9 grains Dicalcium Phosphate, 6 grains Calcium Gluconate and 660 U. S. P. X (1934 Rev.) units of Vitamin D. They are wintergreen flavored, very palatable and may be chewed like candy.

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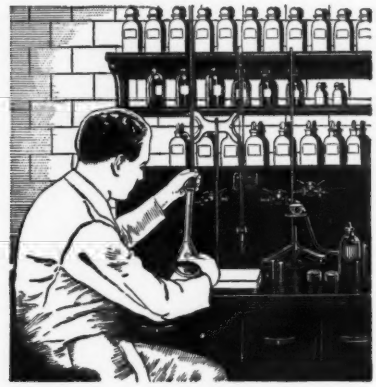

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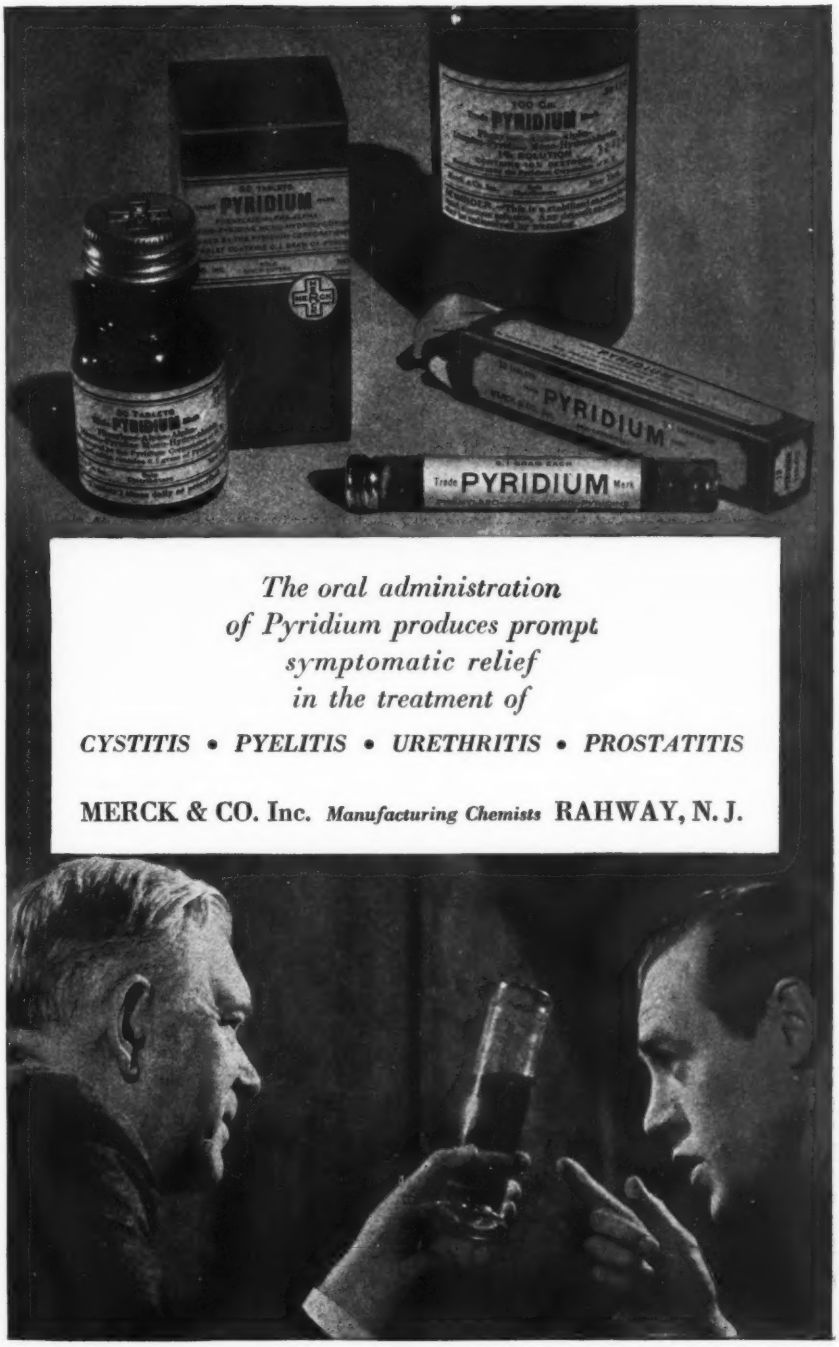

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Continued from page 22

Dr. Elliott Knight Dixon, St. Louis, aged 44, died June 25 of pneumonia.

Dr. Martin Yates, Fulton, aged 83, died April 9 of arteriosclerosis.

Dr. Charles H. Christian, Fulton, aged 74, died recently of arteriosclerosis.

Dr. William T. Mahon, St. Louis, aged 82, died May 13 of pyelitis.

NORTH CAROLINA

The Guilford County Medical Society had as guest speaker at a recent meeting Dr. C. M. Byrnes, of Baltimore, Maryland.

Dr. Grant Fleming, Professor of Public Health and Preventive Medicine at McGill University, Quebec, Canada, was a recent visitor to the Duke University School of Medicine, Durham.

Dr. Paul H. Ringer, Asheville, entertained the members of the Buncombe County Medical Society at a picnic supper at his home on June 17.

Dr. L. R. Staton has located at Murphy for the practice of medicine.

Dr. Robert D. Croom, Jr., has located at Maxton for general practice.

Dr. M. T. Plyler has located at Sanatorium, where he is on the staff of the North Carolina Sanatorium.

Dr. F. V. Taylor, formerly of Stanley, has opened offices in the Independence Building, Charlotte.

Dr. James Cornelius Paff Fearrington, Winston-Salem, and Miss Florence McCanless were married recently.

Dr. Stephen W. Davis, Charlotte, and Miss Kate Jones were married June 8

Dr. Ashley Curtis Norfleet, Tarboro, and Miss Dorothy Edwards were married June 21.

Deaths

Dr. Albert DeKalb Parrott, Kinston, aged 52, died April 19 of chronic myocarditis.

Dr. William Harvey Dixon, Ayden, aged 62, died recently of angina pectoris

Dr. R. B. Killian, Lincolnton, aged 79, died June 27.

OKLAHOMA

The Southeastern Oklahoma Medical Association met at Durant on June 27.

The Southern Oklahoma Medical Association held its quarterly meeting at Sulphur on June 4.

Dr. John F. Park and Dr. Walter J. Dell, both of McAlester, have been doing postgraduate work at the University of Vienna.

Dr. V. H. Barton, McAlester, was recently appointed State Prison physician.

Dr. John I. Gaston, Shawnee, was recently appointed Superintendent of Health of Pottawatomie County.

Dr. W. G. Husband, Hollis, has reopened his hospital after returning from Rochester, Minnesota.

Dr. W. L. Shippey, formerly of Wister, has located in Poteau and will occupy the office of the late Dr. Harrell Hardy.

Deaths

Dr. Thomas J. Colley, Hominy, aged 64, died June 4 of coronary thrombosis.

Dr. Georgia Sizer Orvis, Guthrie, aged 86, died May 13 of chronic myocarditis.

Dr. Frank Robertson, Blackwell, died May 21 of a paralytic stroke.

Dr. G. O. Dunseth, Tulsa, aged 57, died recently of pneumonia.

SOUTH CAROLINA

Dr. William A. Carrigan, Society Hill, has been appointed Health Officer for Darlington County.

Dr. Sedgwick Simons, Georgetown, has been made Health Officer of Beaufort County.

Deaths

Dr. James Baruch Hughey, Kingstree, aged 75, died June 13 of endocarditis.

Continued on page 28

Late Summer and Early Fall

Diarrhea in Infancy

Intestinal disturbances of infants are likely to be as frequent and even more severe now than in early summer.

It is therefore timely to suggest again the following rational and efficient procedure as a means to prevent the development of a serious diarrhea.

Mellin's Food 4 level tablespoonfuls

Water (boiled, then cooled) 16 ounces

Give one to three ounces every hour or two until the stools lessen in number and improve in character.

The mixture may then be strengthened by the gradual substitution of boiled skimmed milk for water until the quantity of skimmed milk is equal to the normal quantity of milk used in the baby's formula. Finally the fat of the milk may be gradually replaced by skimming less and less cream from the milk.

Samples sent to physicians upon request.

Directions for using Mellin's Food are left entirely to the physician.

Mellin's Food Company, Boston, Mass.

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"THE *amino-acid radicals of bovine milk are somewhat different from those of human milk. In general, it may be stated that the infant requires twice as much of the foreign protein as the protein in human milk in order to satisfy its requirements.*"—*Zahorsky.* Synopsis of Pediatrics.

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THE need for higher protein values in the artificial formula to compensate for biological differences between cow's milk protein and the protein of human milk, is voiced by many authorities. The above quotation is a typical one.

In the weak cow's milk dilutions made necessary by digestive limitations during early infancy, this increased protein need is rarely supplied.

The greater digestibility of Dryco permits feedings that supply ample protein throughout the entire nursing period. Moreover, following Nature's own lead in breast milk, modified Dryco feedings supply their highest protein content during early months when growth is fastest and the protein need greatest.

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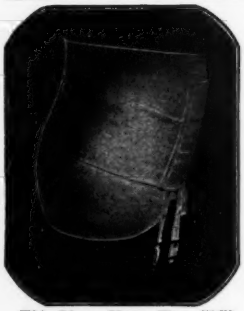

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Continued from page 26

Dr. William Calvin Brice, York, aged 35, died June 11 of heart disease.

Dr. Carl Atkins Forster, Columbia, aged 56, died recently of coronary occlusion.

TENNESSEE

Dr. Joe T. Smith, Knoxville, has been elected President of the Tennessee Pediatric Society.

Dr. William W. Potter, Knoxville, has been elected President of the Tennessee Academy of Ophthalmology and Otolaryngology.

Dr. John W. L. Cooper was recently appointed Health Officer of Chattanooga, succeeding Dr. Frederick C. McIsaac.

Dr. Eugene B. Elder, formerly Superintendent of Knoxville General Hospital, has been appointed Superintendent of Erlanger Hospital, Chattanooga. Dr. Elder will be succeeded at the Knoxville Hospital by Dr. Andrew Smith, former Chief of Staff.

Dr. J. A. P. Shields, formerly of Hartford, has accepted a position at Rainbow Springs, North Carolina.

Dr. Dean DeWitt Lewis, Baltimore, was guest speaker at the Chattanooga Surgeons' Club on July 9.

Dr. James E. Paullin, Atlanta, President of the Georgia Medical Association, and Dr. William Thornwall Davis, Washington, District of Columbia, were speakers before the Tennessee Valley Medical Association at Knoxville in June.

Dr. J. A. Crisler, Sr., and Dr. Harley Harris, Memphis, were guest speakers before the Northeast Mississippi Thirteen Counties Medical Societies recently.

Dr. and Mrs. J. F. Adams, Woodbury, entertained the doctors of Cannon, Smith and Rutherford Counties and their wives at a picnic supper.

Dr. Gideon W. Stones, Knoxville, has been taking postgraduate work in obstetrics and gynecology at Washington University, St. Louis.

Dr. Robert J. Bogan and Miss Evelyn Frances Summers, both of Memphis, were married June 1.

DEATHS

Dr. Martin Luther Bradley, Springfield, aged 75, died June 26 of chronic valvular heart disease.

Dr. William H. Longmire, Elizabethton, aged 44, died May 16 of hypertensive heart disease and coronary occlusion.

Dr. S. E. Parrott, Cordova, aged 61, died recently.

Dr. M. L. Bradley, Springfield, aged 78, died June 26 following an illness of six months.

TEXAS

The American Society of X-Ray Technicians held its annual meeting May 20-24 at Dallas.

Dr. Thomas N. Goodson, San Antonio, was recently appointed Health Officer of Bexar County.

Dr. Lawton C. Biggers, Bonham, has been appointed Health Officer of Fannin County.

Dr. J. J. Cappleman, Honey Grove, has been named City Health Officer, filling out the unexpired term of Dr. J. C. Joiner, deceased.

Dr. Hubert S. Jackson, San Antonio, and Dr. William P. Harrison, Teague, were recently appointed to the State Board of Health.

Dr. Walter Sievers has been appointed City Health Officer of Gonzales.

Dr. W. B. Carrell, Dallas, was elected to membership in the International Orthopedic Association at its annual meeting in London.

Dr. A. C. Scott, Sr., Temple, has been awarded the degree of Doctor of Laws by the Trinity University, Waxahachie.

Dr. W. S. Barcus, Fort Worth, has returned after taking a month's postgraduate work in the East.

Dr. Horace E. Duncan, Dallas, following nine months of study in health work at Johns Hopkins University, Baltimore, has resumed his official duties as Health Officer of Dallas County.

Dr. Byford H. Denman has been in New York, where he took postgraduate work at the New York Polyclinic Medical School and Hospital.

Dr. Herbert Merz, Alvin, is at the Mayo Clinic for the summer.

Dr. N. B. Daniel, formerly of Texarkana, Arkansas, has moved to Mercedes.

Dr. M. O. Dart, formerly of Bonham, has moved to Denver, Colorado.

Dr. Edwin E. Connor has moved from Pampa to Baytown.

Continued on page 30

Breast Milk from the Normal Mother is the Ideal Food for the Human Infant

That is Why

S. M. A. Is Made to Resemble BREAST MILK

in percentages of carbohydrate, protein, fat and total salts (ash) content, and why even the chemical and physical constants of the fat in S. M. A. are like those of breast milk fat.

S.M.A. is a food for infants — derived from tuberculin tested cows' milk, the fat of which is replaced by animal and vegetable fats including biologically tested cod liver oil; with the addition of milk sugar and potassium chloride;

altogether forming an antirachitic food. When diluted according to directions, it is ***essentially similar to human milk*** in percentages of protein, fat, carbohydrates and ash, in chemical constants of the fat and in physical properties.

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RACINE, WIS.

Continued from page 28

Dr. T. G. Edwards has moved from Dublin to Cross Plains.
Dr. Herbert E. Hipps has moved from Houston to Marlin.
Dr. L. C. Kopecky has moved from Yoakum to San Antonio.
Dr. W. T. Malone has moved from Navasota to Kerrville.
Dr. T. A. Moore has moved from Eliasville to Jermyn.
Dr. Everett C. Fox, Dallas, and Miss Ella Gertrude Boyce were married June 5.
Dr. William W. Shortal and Miss Minnie Bell Graham, both of Dallas, were married June 14.

Deaths

Dr. James Granger Poe, Dallas, aged 62, died June 15 of coronary occlusion.
Dr. Joshua Zadok Sexton, Waco, aged 63, died June 3 of cerebral hemorrhage and hypertension.
Dr. Richard Henry Moers, Houston, aged 49, died May 24 of embolism and coronary thrombosis.
Dr. Mary J. Whittet, Anchorage, aged 81, died May 24.
Dr. Frank P. McLaughlin, Austin, aged 80, died May 25.
Dr. C. I. Holt, Olton, aged 75, died recently of pneumonia.

VIRGINIA

The Halifax County Medical Society has elected the following officers for the coming year: Dr. Lloyd Eastlack, South Boston, President; Dr. L. H. Hoover, Clarksville, Vice-President; Dr. William C. Brann, South Boston, Secretary-Treasurer.

The Lee County Medical Society elected the following officers for the coming year: Dr. W. J. Innes, Pennington Gap, President; Dr. J. B. Muncy, Pennington Gap, Secretary-Treasurer.

Dr. O. C. Brunk, Richmond, has been named Police Surgeon, succeeding the late Dr. B. L. Hillsman.

Dr. Karl S. Blackwell, Richmond, has been elected President of the Medical College of Virginia Alumni Association.

Dr. Emory Hill, Richmond, has been elected President of the Virginia Society of Otolaryngology and Ophthalmology.

Dr. Guy R. Harrison, Richmond, was recently reappointed by Governor Peery as a member of the State Board of Health.

Continued on page 32

THELESTRIN
OVARIAN FOLLICULAR HORMONE

A standardized preparation of the ovarian follicular hormone for intensive ovarian therapy by hypodermic injection.

Indicated in the treatment of scanty menstrual flow, amenorrhea and the vasomotor and nervous symptoms of the menopause.

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Readily ASSIMILABLE FAT -- an essential in FEEDING THE PREMATURE

IN a recent study of fat metabolism in infants, Holt, Tidwell and Kirk* report that olive oil showed the highest percent retention (95.1%) of all fats studied but one, olein (97.5%). These authors found the fat of SIMILAC (which is 20% olive oil) showed a better percent retention (92.6%) than butter fat (88.9%)—and as high a retention as breast milk fat (92.4%).

To quote these authors—"the differences in fat retention on these various fats as shown on normal infants are not great; for the normal infant it is probably immaterial whether he absorbs 85% or 95% of his fat intake. It seemed possible, however, that *in subjects who have difficulty in fat assimilation, such as premature infants, the observed small differences might become large differences. A few observations made on premature infants and twins have borne this out*—."

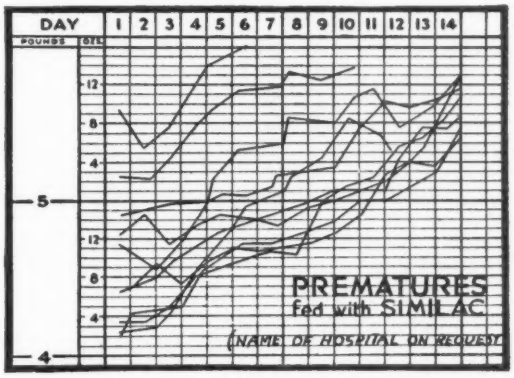
The observations referred to covered only three prematures fed on different fats, but showed an average of 78.4% retention for olive oil as compared to only 52.5% retention for butter fat.

*Holt, Tidwell and Kirk, Studies on Fat Metabolism in Infants—Acta Pediatrica, Vol. XVI, 1933.

SIMILAC

has given noticeably good results in feeding the premature infant. One of the reasons lies, as here pointed out, in the composition of its fat. Another reason is its consistently zero curd tension. The finer the curd the greater the surface area. The greater the surface area the more exposed are the fats, carbohydrates, proteins and salts to the digestive enzymes. Result . . . the food substances are more quickly and readily utilized.

SIMILAC is made from fresh skim milk (casein modified) with added lactose, salts, milk fat, and vegetable and cod liver oils.



The fact that SIMILAC is well assimilated by the immature digestive tract of the premature indicates how entirely suitable it is for all those infants who are deprived of breast milk.

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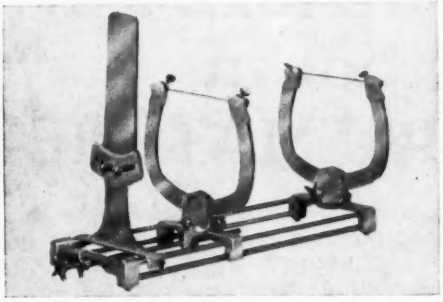

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Continued from page 30

Dr. Thomas F. Wheeldon, Richmond, has been elected President of the Harvard Club of Virginia.

Dr. John A. Gibson, Leesburg, has been elected President of the Leesburg Rotary Club.

Dr. Harry Golston, Roanoke, has been at Harvard University Medical School, Boston, taking postgraduate work in diseases of the heart.

Dr. Richard S. Herring, Richmond, is in Vienna, where he is taking postgraduate work at the University of Vienna.

Dr. William S. Lloyd, formerly of Richmond, has located at Bumpass for general practice.

Dr. Paul Q. Daniel, formerly of Deltaville, has located at Highland Springs and has taken over the home and practice of the late Dr. James T. Leftwich.

Dr. Samuel H. Garst, formerly of Baltimore, Maryland, has located in Staunton for the practice of surgery and orthopedics.

Dr. C. G. O'Brien, formerly of Richmond, has located at Appomattox for general practice.

Dr. William R. Pretlow, formerly of Richmond, has located at The Plains for general practice.

Dr. J. Paul Kent, formerly of Roanoke Hospital, has located on Williamson Road.

The Fauquier County Medical Society elected the following officers for the coming year: Dr. Richard Mason, The Plains, President; Dr. George H. Davis and Dr. J. E. Knight, both of Warrenton, Vice-Presidents; Dr. Martin B. Hiden, Warrenton, Secretary-Treasurer.

Dr. James Porter Baker, Jr., and Miss Louise Herbert Rogerson, both of Richmond, were married June 8.

Dr. Virgil Leonidas Kelly, Charlottesville, and Miss Alberta Worrell were married June 11.

Dr. Charles Morris Nelson and Miss Charlotte Mercer Purcell, both of Richmond, were married July 16.

Dr. Fletcher Johnston Wright, Jr., Petersburg, and Miss Martha Jeanette Andrews were married June 29.

Dr. Charles Roy Hoskins, Jr., and Miss Virginia Eubank Segar, both of Saluda, were married July 1.

Dr. Oliver Allison Ryder, Alexandria, and Miss Elinor Temple Hill were married July 6.

Dr. Lewis Littlepage, Jr., Norfolk, and Miss Eleanor Mattingly were married June 29.

DEATHS

Dr. Albert C. Stephens, Barren Springs, aged 65, died May 14 of angina pectoris.

Dr. Calvin Graham Dold, Buena Vista, aged 65, died June 22 of coronary thrombosis.

WEST VIRGINIA

Dr. John Royal Moore, Surgeon-in-Charge of the Shriners' Orthopedic Hospital, Philadelphia, was the guest speaker at the meeting of the Fayette County Medical Society on June 21.

The Ohio County Medical Society elected the following officers for the coming year: Dr. Russell Bond, Wheeling, President; Dr. Robert J. Armbrecht, Wheeling, Vice-President; Dr. W. M. Sheppe, Wheeling, Secretary; Dr. James K. Stewart, Wheeling, Treasurer.

Dr. Charles Y. Moser, Terra Alta, has succeeded Dr. Edward R. Davies, Kingwood, as Health Officer of Preston County, Dr. Davies having resigned to take a similar position in Baltimore.

The Monongalia, Marion and Harrison County Medical Societies, in joint session June 4, had as guest speakers Dr. William S. Love, Jr., and Dr. Charles Reid Edwards, Baltimore, Maryland.

The Tyler-Wetzel Counties Medical Society had as guest speaker at a recent meeting Dr. Joseph H. Barach, Pittsburgh.

The Preston and Monongalia County Medical Societies, in joint session, had as guest speaker recently Dr. John Strieder, University of Michigan, Ann Arbor.

Dr. S. L. Rucker, formerly of Moneta, Virginia, has located at Elkview for the practice of medicine.

Dr. Wycliffe Charles Jackson, Raysal, and Miss Mary Stuart McCallum were married June 1.

Dr. Fletcher Johnston Wright, Jr., Princeton, and Miss Martha Jeanette Andrews were married June 29.

DEATHS

Dr. James Angus Monroe, Wheeling, aged 82, died May 30 of coronary occlusion.

Dr. John Wallis Walker, Winona, aged 78, died recently of cerebral hemorrhage.

Dr. Edward L. Armbrecht, Wheeling, aged 65, died July 10 from a heart attack.

Dr. A. J. White, Charleston, aged 48, died July 1.

NO ADDED TAX

on the Invalid's Stomach with

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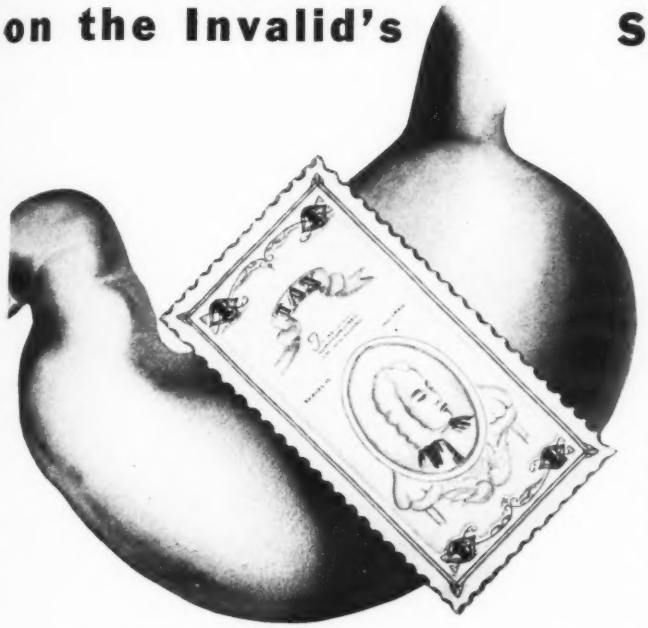

When digestive tolerance is low, following illness or operation, here is a safe way to meet the increased food requirements of convalescence.

In everyday dishes, such as soups and cereals, simply include KLIM Powdered Whole Milk.. Each tablespoonful of KLIM adds 42 extra calories with slight change in flavor, consistency or bulk.

KLIM is so digestible, so easily assimilable, that when used to fortify staple dishes it throws little extra burden on the weakened stomach. Most important, patients will welcome the wide variety of foods that can be made with KLIM. So different from the monotony of highly flavored, rapidly cloying "invalid drinks."

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"Hurry, Jimmie, or you'll be late for school again. Mother forgot to set the alarm clock. Please don't DAWDLE like that. Here, take your bun and eat it on your way to school. HURRY, darling, teacher will have a fit! PLEASE hurry!"
THE LAST-MINUTE BREAKFAST

THE milk is the best item in this child's hurried, harried, worried breakfast, but milk alone is inadequate. The simple replacement of the bun or roll by Pablum would, with added milk, give the child a better-constituted and more nourishing meal on which to start the day right at school. Pablum can be prepared appetizingly, in a few seconds' time, without cooking.

"Going to school on an empty stomach"

—not because his parents are poor or illiterate, but because his mother didn't allow sufficient time for an adequate, nourishing morning meal.

This scene occurs every morning in thousands of homes, and many a school child is a poor scholar because of a poor breakfast.

For little boys and girls* whose mothers don't get up early enough in the morning, or who can't figure time accurately, a good, nourishing, well-constituted, economical and *quick* morning meal is:

Orange Juice or Tomato Juice	**qs**
Pablum & milk or cream	**qs**
Sugar	**qs**
Capsule, Mead's Viosterol in Halibut Liver Oil	**I**
More Milk	**qs**

Such a breakfast supplies important amounts of all the following essential nutritional requirements: **Protein,✓ Fat,✓ Carbohydrate.✓ Vitamins: A, B, C, D, E, G.✓ ✓ Minerals: Calcium, Phosphorus, Iron, Copper, Etc., Etc.✓✓✓ Calories.✓**

PABLUM can be prepared in less than a minute and does away with pots and pans and endless overnight and early-morning cereal cookery and drudgery. Simply add milk or water of any desired temperature and serve with cream, salt and sugar.

Pablum (Mead's Cereal thoroughly pre-cooked by a patented process) consists of wheatmeal, oatmeal, cornmeal, wheat embryo, alfalfa leaf, beef bone, brewers' yeast, iron salt, sodium chloride. Mead Johnson & Company, Evansville, Ind.

*and perhaps also for their fathers who have to gulp a one-minute breakfast before going to work.

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Proved Facts Concerning DEXTRI-MALTOSE

Continued down from 1911

1931

"Dextri-maltose has been substituted for lactose not infrequently, when the tolerance for the latter continues low."—*J. H. West: Low fat, high starch, evaporated milk feeding for the marasmic baby, Arch. Pediat. 48:189-193, March 1931.*

1931

In the feeding of prematures, "As soon as there is a hesitation in the gain in weight, dextrimaltose No. 1 is substituted for the dextrose, in the same amount in the mixture, with almost invariably a gain in weight."—*F. B. Jacobs: Relation of irradiated food substances and ergosterol versus cod liver oil in childhood nutrition, Pennsylvania M. J. 35:164-167, Dec. 1931.*

1931

"It should be remembered that more than 6% of lactose may cause diarrhoea. If a higher percentage of sugar be required it is better to replace it by dextri-maltose, such as Mead's Nos. 1 and 2, where the maltose is only slightly in excess of the dextrins, thus diminishing the possibility of excessive fermentation."—*W. J. Pearson: Common practices in infant feeding, Post-Graduate Med. J. 6:38, 1930: abst. Brit. J. Dis. 28:152-153, April-June 1931.*

1931

"Malt sugar is indicated in the following conditions: (1) in severe atrophies, (2) in cases of fat indigestion before the atrophic stage is reached, (3) in cases where there is slight curd indigestion, indicated by some vomiting and slow gain in weight, (4) in cases where excessive intestinal fermentation is manifested by gas and colic."—*F. T. Proudfit: Dietetics for Nurses, Macmillan Co., New York, 1931, p. 174.*

1932

"After the preliminary short period of starvation, protein milk should be used. . . . When the diarrhoea has been sufficiently checked, dextri-maltose may be added and gradually increased until from 4 to 6 tablespoons are being used."—*W. L. Denney: Acute nutritional disturbances of infancy, Univ. West. Ontario M. J. 2:132-137, April 1932.*

1932

In cases of diarrhea, "For the first day or so no sugar should be added to the milk. If the bowel movements improve carbohydrates may be added. This should be the one that is most easily assimilated, so dextri-maltose is the carbohydrate of choice."—*W. H. McCaslan: Summer diarrheas in infants and young children, J. M. A. Alabama, 1:278-282, Jan. 1932.*

1932

"The malt preparations are preferred generally because less easily fermented."—*O. N. Torian: The evolution and present-day status of infant feeding, J. Indiana St. M. A. 25:77-80, Feb. 1932.*

1932

In diarrhea, "The sugar is added gradually as conditions admit, some sugar other than milk sugar or cane sugar being used, preferably dextrin and maltose."—*H. E. Small: Diarrhoea in bottle-fed infants, J. Maine M. A. 12:152-158, Jan. 1932.*

1933

"Some of the Dextrin Malt mixtures are preferred by most clinicians to the other sugars."—*T. J. Marshall: The treatment of diarrhea in infants, Kentucky M. J. 31:60-62, Jan. 1933.*

1933

"However, it is in the correction of nutritional disturbances that a great difference in the action of sugars may be seen. The addition of maltose-dextrin preparations, malt soups or cereals will prove most efficacious in clearing up a simple dystrophy. As has been already noted, a greater quantity of dextrin, starch or malt may be ingested with a lesser amount of fermentation than any of the other sugars, and this is exactly what is needed for the treatment of the condition; to add more sugar of a type not easily fermentable. They are safer because they can be added in greater quantity, with less danger of excessive fermentation. They may be added up to from 5 to 10% of the total milk formula."

"The treatment of dyspeptic diarrhea must aim at measures to prevent the excessive fermentation."

"Sugars must be added gradually, and those only with the least tendency to fermentation, for it is an excessive fermentation which we are attempting to combat. Malt-dextrin preparations, wheat flour, corn starch, rice flour, and oatmeal may be cautiously added to the diet."—*A. F. Abt: The use of carbohydrates in the diet and treatment of infants, Nebraska State M. J. 18:241-246, July 1933.*

1933

"Dextri-Maltose can be fed in larger amounts than other sugars, because its assimilation limits are higher. Cane or milk sugar can be given in the proportion of three gm. per pound of body weight per day, added to the milk sugar in the milk. Dextri-Maltose can be given in amounts of four gm. per pound of body weight."

"A very frequent cause of underfeeding results from the improper treatment of diarrhea. . . . . One of the greatest advances made in the science of infant feeding was the development of protein milk by Professor Finkelstein and the use of buttermilk or lactic acid mixtures. The great advantage of being able to feed the infant with fermentative diarrhea a food containing 12 calories to the ounce, like protein milk, after only one day on a starvation diet, is apparent. In addition, the further advantage of being able to safely add a carbohydrate like Dextri-Maltose No. 1 or No. 2 to the protein milk within a few days, enables one to gradually bring the infant up to its basal needs in a short time. When protein milk was first used, carbohydrate additions were advised against with the result that many children on it went into a state of collapse. The suggestion of Dr. Alan Brown of Toronto, Canada, that Dextri-Maltose be added to protein milk, was of great value. Too many practitioners still use protein milk for prolonged periods without adding carbohydrate; it must be emphasized that regardless of the condition of the stools, carbohydrate must be added to protein milk within a reasonable time in order to avoid collapse."—*G. J. Feldstein: Underfeeding of infants and children, Arch. Pediat. 50:297-306, May 1933.*

1933

In the treatment of simple atrophy, "The carbohydrate of choice, because of its low fermentative power, appears to be a mixture of dextrose and maltose, the so-called 'Dextro-maltose'."

"In the mild and moderately severe cases of atrophy the giving of adequate amounts of lactic acid milks with added dextro-maltose will enable the infant to gain."—*A. Maitland-Jones: Diseases of Infancy and Childhood (edited by L. G. Parsons and S. Barling), Oxford University Press, New York, 1933, vol. 1, pp. 374 and 376.*

1933

"In choosing the carbohydrate, consideration must be given to the ease of digestion and absorption, the degree to which it is fermented in the gastro-intestinal tract, and the amount of irritation that it causes. From these standpoints the sugars composed largely of dextrin and maltose mixtures seem to more nearly meet the needs of the average infant, though the others also can often be used with good results, and in certain cases may be especially indicated."—*J. B. Stone: Infant feeding, Virginia M. Monthly, 60:176-182, June 1933.*

1933

"The dextrins are produced during the hydrolysis of starch through the action of certain ferments; they are in turn converted to maltose, and finally to glucose as hydrolysis is completed. The dextrins, though slowly digested, do not readily ferment, and are therefore less likely to cause diarrhoea than most other forms of carbohydrate. Maltose is easily digested, though readily fermentable. The malt preparations vary in their laxative properties, which depend upon the relative proportions of dextrins and maltose present in them. The preparation known as 'Dextri-maltose' is very satisfactory and convenient for use. Its value lies in its combination of carbohydrates (approximately maltose 51 per cent., dextrin 41 per cent.); it is easily assimilated, and less fermentable than either maltose or cane sugar alone. It therefore has no special tendency to cause diarrhoea, and is very useful in the case of young or weakly infants."—*K. H. Tallerman: Diseases of Infancy and Childhood (edited by L. G. Parsons and S. Barling), Oxford University Press, New York, 1933, vol. 1, p. 177.*

1933

"Dextrin is very little fermented by the bacteria of the intestinal flora, but is easily converted into maltose by enzyme action. This conversion is not extremely rapid, so that there is never a surplus of readily fermentable carbohydrate available, which is of advantage in the prevention and treatment of diarrheal conditions. Consequently, it is believed that more carbohydrate may be given without danger of producing diarrhea in the form of mixtures of dextrin-maltose and dextrose, than in the form of lactose, sucrose, or pure dextrose."—*A. F. Abt: The use of carbohydrates in the diet and treatment of infants, Nebraska St. M. J. 18:206-209, June 1933.*

Continued down to 1934

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"Sleep, mildest of all the gods, thou art thyself sweet peace of mind, a soothing balm, an alien to care, and brightest rest and strength to mortals worn and weary with the toils of life."

—OVID

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